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DEPARTMENTS

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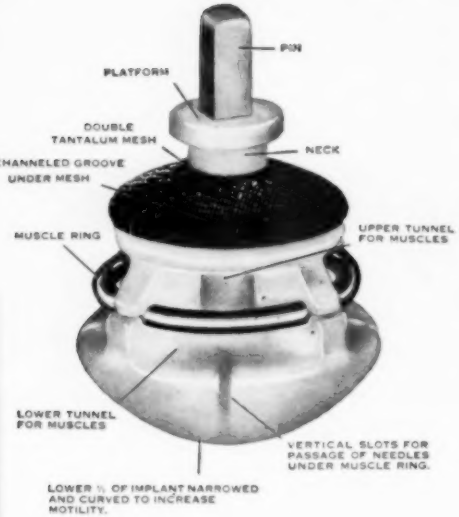

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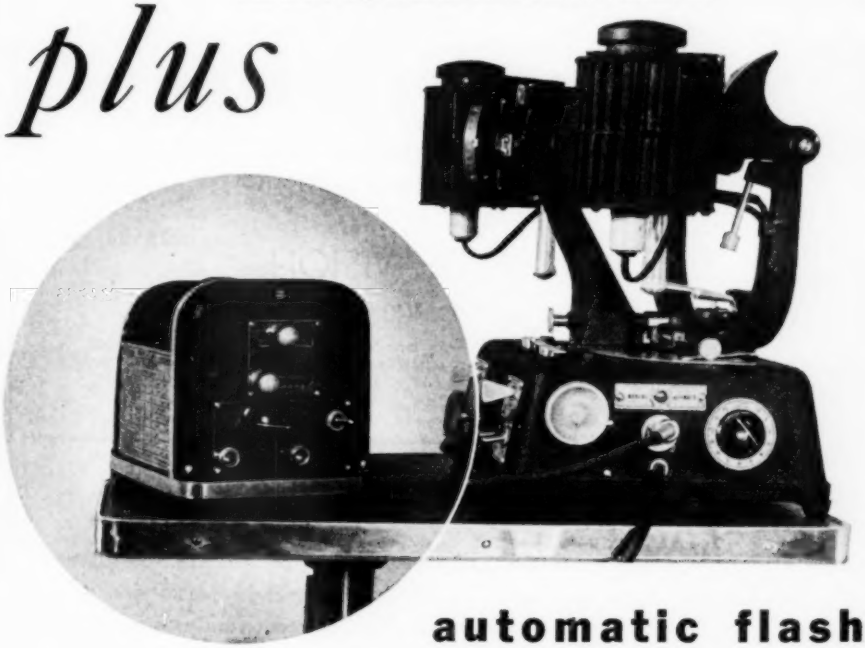

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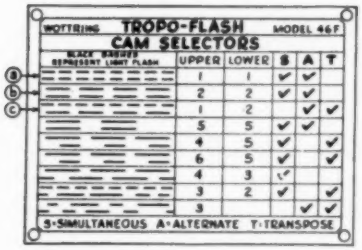

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Chromatic aberration is usually associated with vision through the bifocal segments of fused bifocals. Occasionally, however, this aberration is noticed through the distance portion of lenses as in the case we have this month.

Mrs. S. is a monocular cataract case. She has excellent vision in the operated eye and practically no vision in the other eye. The aphakic eye requires A + 11.50 on + 3.00 axis 175 with A + 3.00 add for reading.

This correction resulted in excellent near and distance vision, but the patient complained at once that everything she looked at was decidedly tinged with blue. Surprisingly the vision through the distance part of the lens was just as blue as the vision through the segment.

An examination of the lens showed no grinding defects. The surfaces were well polished, the correction and axis were perfect but at the point through which the patient looked, when her eyes were directed straight ahead, there was a 6 degree prism base out.

Before making a new lens for Mrs. S. we neutralized this base out prism by placing a 6 degree prism over the lens base in. The patient at once reported that the blue cast was gone.

A new lens perfectly centered was made and the patient is satisfied.

Cases like this point up the fact that proper centering is of great importance in monocular cases. Too often off-centered lenses are passed on to the patient with the false justification that since only one eye was being used the centering was of no importance.

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 32 JULY, 1949 NUMBER 7

THE DIAGNOSIS OF ORBITAL TUMORS BY ANGIOGRAPHY*

ANTONIO GRINO, M.D. AND EDWIN BILLET, M.D.
New York

The differential diagnosis between intraorbital and certain intracranial tumors constitutes a difficult problem which is the concern of ophthalmologists as well as neurosurgeons.

Unilateral exophthalmos and accompanying unilateral loss of vision manifested either by diminution in central acuity or irregular constriction of the visual field on the affected side, or both, may be accepted as the signs par excellence of orbital tumor. These signs, which attract the early attention of the patient as well as that of the clinician and which are pointing, so to speak, toward the site of the lesions, may, however, be deceiving, since they are also present in certain cases of intracranial growth (sphenoidal wing meningioma) and in cases of intracranial tumor with orbital extension. Furthermore, since intraorbital tumors may not be accompanied by proptosis (as has been shown by Reese[7]), the error of mistaking an orbital tumor for an intracranial growth, or vice versa, is neither infrequent nor avoidable, even for the most experienced diagnostician.

Because errors in diagnosis may lead to submitting the patient to an insufficient or unnecessary intracranial exploration, the importance of accurate differential diagnosis cannot be overemphasized.

Roentgen examination of the skull, orbits, and optic foramina is used routinely. Aspiration biopsy, surgical biopsy, and retrocular injection of air may also be of some help in making a diagnosis. In many cases, pneumo-encephalography is resorted to, after using some or all of the foregoing methods. Nevertheless, even with the use of all these diagnostic procedures, a great deal of doubt may still be cast upon the diagnosis.

The purpose of this paper is to demonstrate that intraorbital tumors can be visualized by means of cerebral angiography. To our knowledge, this particular use of cerebral angiography has not been reported to date.

Two cases of angiographic visualization of intraorbital tumors will be presented, along with a third case which showed signs of intraorbital tumor but definite angiographic evidence of intracranial growth.

Since the description of cerebral angiography by Egas Moniz,[6] this method has been used for the diagnosis of intracranial lesions, although it was, until recent years, generally limited to the diagnosis of suspected intracranial vascular malformations.

That an intracranial tumor also could be visualized by the filling of its vessels had already been shown by Egas Moniz,[6] Trias,[8] and others; but the lack of a safe, although adequate, contrast medium limited the clinical application of this method.

Strontium iodide, sodium iodide, and sodium bromide, which were used in the beginning, were discarded when thorotrast appeared. However, while thorotrast does not produce the same harmful reactions that the inorganic halogen derivatives do, its radioactivity and its persistence in the organism

* From the Neurosurgical Service of Montefiore Hospital and St. Vincent's Hospital, and from the Ophthalmological Service of Montefiore Hospital.

for years limit its use. With the use of diodrast (introduced by Gross[4]) or similar compounds which are soluble, nontoxic, and easily eliminated from the organism, these objections have been overcome. A further advance, also facilitating the routine application of angiography, is the development of the percutaneous technique described and used for the first time by Loman and Myerson.[5]

Since the details of the percutaneous technique, which we use exclusively whenever possible, will be published elsewhere by one of us, we shall give here only a general resume of it.

Under local anesthesia, we insert into the common carotid artery a three-piece Cournand needle which is made 3.5 inches long and 17 gauge especially for us.* The advantages of this needle will be discussed by one of us at length in the forthcoming paper. Ten cc., or less, of 35-percent diodrast are injected at the highest possible speed (average two seconds).

A system for rapid changing of the X-ray plates is indispensable in order to obtain the three phases of the angiogram, namely, the arteriogram, the capillarogram, and the venogram. This is easy to understand if one bears in mind that the contrast medium passes from the arterial to the venous system in five seconds. Because the image of a tumor will be better demonstrated in the capillary and the venous phases, due to the relative abundance of the capillary and venous supplies, it is imperative to obtain all three phases, which, moreover, are of complementary diagnostic value.

In the few cases in which we are not able to insert the needle into the carotid by the percutaneous method, we use the open method. This is done by a small (1 inch) cutaneous incision on the supraclavicular region between the heads of the sternocleidomastoid muscle. After retraction of the edges of the cutaneous wound, the common carotid artery is easily identified by palpation, the carotid sheath is dissected to a length of 1 or 1.5 inches, and a piece of vaselined tape is passed underneath the vessel. The wound is closed with through-and-through sutures which are not tied until the angiography is performed. We have not had any harmful reaction in the numerous patients who have been subjected to this test. When performed percutaneously, angiography may be an ambulatory procedure.

* This needle can be purchased from Becton & Dickinson.

Case Reports

Case 1

History. A. I., a 34-year-old white woman, was admitted to the neurosurgical service of Montefiore Hospital on November 5, 1947, with a 5-year history of slowly progressive proptosis of the right eye. Six months prior to admission, A. I. became aware of a decrease in vision in the right eye, and her vision in this eye worsened progressively. She began having headaches five weeks prior to admission. There were no other neurologic symptoms. Her past history was significant only in the fact that she had had an episode of rheumatic fever at the age of 14 years and again a year prior to admission.

Physical examination. The general physical examination revealed a well-developed and well-nourished woman. There were no abnormalities. The neurologic examination revealed normal gait, station, and coördination. All reflexes were active and equal. Sensation was intact and there was no motor weakness. The Babinski test was negative. All the cranial nerves were unimpaired except the optic nerve.

Ocular examination. Dr. Max Chamlin saw this patient the day after admission and contributed the following note and visual field studies.

Corrected visual acuity: O.D., 15/100−1; O.S., 15/15. With the Hertel exophthalmometer on a 100 base line, the right eye

measured 27 mm. and the left eye measured 19 mm. (fig. 1). The proptosis of the right eye was apparent grossly. There was no bruit and no pulsation of the right globe. The right globe could be pressed back into the orbit with no more than normal resistance. Pupillary responses to light and convergence-accommodation were prompt and equal in both eyes. There were no palsies of the extraocular muscles. The left fundus appeared normal and the right fundus showed temporal pallor of the nervehead.

Visual field studies of the right eye, with 3/330 white, 1/330 white, and 1/2,000 white, showed peripheral contraction, more marked temporally, with hemianopic characteristics. The left field was within normal limits to all visual angles.

It was the impression of Dr. Chamlin that the visual fields suggested interference with the right optic nerve retrobulbarly extending rather far back toward the chiasmal area (figs. 2 and 3).

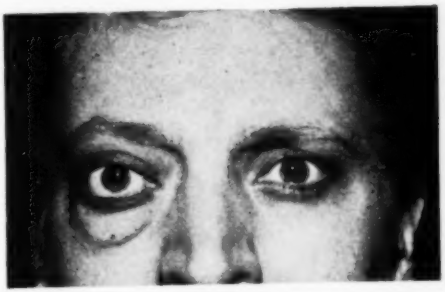

Fig. 1 (Grino and Billet). *Case 1.* Showing proptosis of the right eye.

Laboratory data. Urine was within normal limits. Blood hemoglobin was 13.5 gm.; R.B.C., 4.62 million; W.B.C., 6,600, with essentially normal differential; B.U.N., 14.9 mg. percent; blood sugar, 90 mg. percent; serum albumin, 4.4 mg. percent; serum globulin, 2.6 mg. percent; B.M.R. was −11.

Electrocardiography revealed a normal sinus rhythm. Electro-encephalography revealed a minor degree of change from the

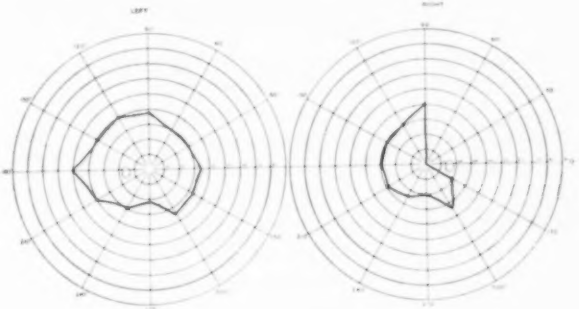

Fig. 2 (Grino and Billet). *Case 1.* Visual fields on November 6, 1947, for 1/330 white. Visual acuity in the right eye was 15/100+1; in the left eye, 15/15.

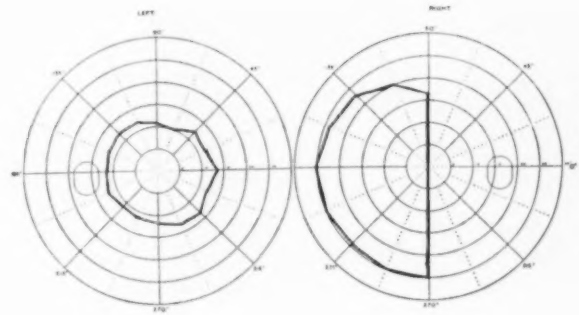

Fig. 3 (Grino and Billet). *Case 1.* Visual fields on March 15, 1948, for: R.E., 20/2,000 red; L.E., 1/2,000 white. Visual acuity: R.E., 15/70; L.E., 15/15.

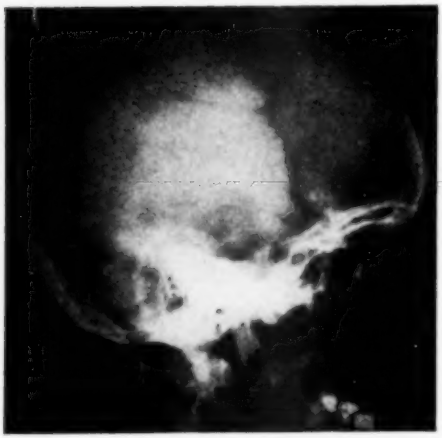

Fig. 4 (Grino and Billet). *Case 1.* Right lateral roentgenogram. The slight bony alterations can be appreciated only in stereoscopic view.

electrical normal in an activity grossly influenced by the patient's drowsiness.

X-ray examination of the chest was within normal limits. X-ray examination of the skull revealed that both the superior orbital plate and the greater wing of the sphenoid consisted of denser but not apparently thicker bone on the right side; the optic foramina appeared normal (fig. 4).

On November 10, 1947, 18 cc. of air were injected behind the right globe. No resistance was met until about 16 cc. were injected. As soon as the injection was completed, there developed marked ecchymosis of both upper and lower lids and of the periorbital skin (fig. 5).

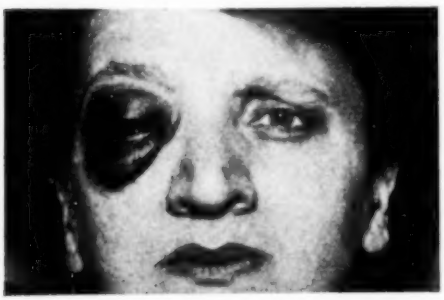

Fig. 5 (Grino and Billet). *Case 1.* Showing marked ecchymosis after air injection.

X-ray examination of the orbits after air insufflation in the postero-anterior view did not disclose any air pockets. In the lateral position there was a very thin layer of air retrobulbarly in the superior portion of the orbital cavity. No tumor was outlined.

On November 12, 1947, a right common carotid arteriogram under general anesthesia was done percutaneously by one of us.

The venograms show a somewhat rounded density, measuring approximately 25 cm. in diameter, within the region of the right orbit posteriorly. A less well-defined density with rather indefinite borders is visible intracranially just above the lesser wing of the sphenoid on the right. These densities were not visible on the preliminary skull films and probably represent capillary circulation in neoplastic masses.

The arteriogram also reveals the just described masses in the region of the right orbit and behind the right lesser wing of the sphenoid. The retro-orbital density is rather poorly defined.

Conclusion. The changes described indicate the existence of a tumor mass in the right orbit, extending possibly retro-orbitally. The intraorbital portion of the mass appears to be highly vascularized (fig. 6). The patient was discharged from the hospital on November 17, 1947, with the diagnosis of retrobulbar tumor of the right eye, probably a hemangioma.

She received roentgen therapy—a total of 16,600r. over a period of one month, from November 28, 1947 to December 29, 1947—through right anterior and lateral portals to the right orbit. Repeated checks of visual acuity, visual fields, and Hertel exophthalmometer readings showed (February 10, 1948) no change in her ocular status.

Ocular examination on June 22, 1948, six months after X-ray treatment, revealed a marked improvement in visual acuity: O.D., from 15/100 to 15/40. There was no recession of the exophthalmos and no change in the fundus picture in this eye. Visual field study with 20/2,000 red re-

Fig. 6 (Grino and Billet). *Case 1.* Right carotid angiography. Venogram showing tumor pointed by arrows. Arrow 2 shows the intracranial extension.

vealed macular sparing to account for the improved acuity (fig. 7).

CASE 2

History. O. H., a 55-year-old white man, had had a left nephrectomy about 8 years ago for painless gross hematuria. Four years ago, the patient developed blurring of vision and proptosis of the left eye. At that time, Dr. Davidoff performed a left exploratory frontal craniotomy. No tumor was found and a decompression was done, including orbital decompression.

For some time, the patient felt better but blurring and proptosis recurred. About one year ago at the Medical Center, a biopsy

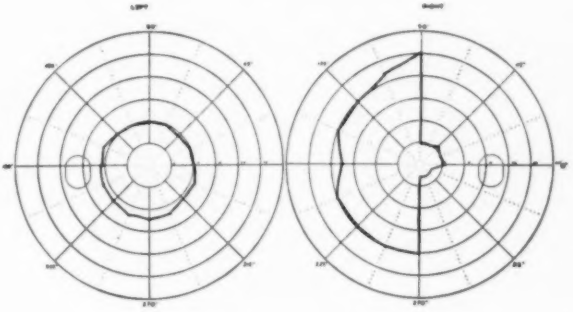

Fig. 7 (Grino and Billet). *Case 1.* Visual fields on June 22, 1948, after roentgen therapy: R.E., 20/2,000 red; L.E., 1/2,000 white. Visual acuity: R.E., 15/40; L.E., 15/15.

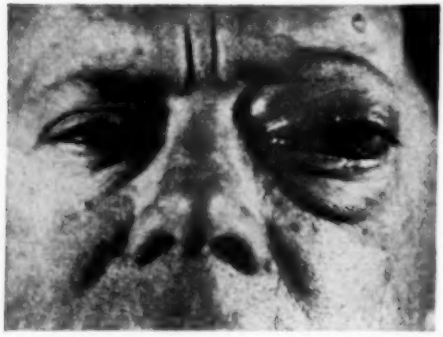

Fig. 8 (Grino and Billet). *Case 2.* Demonstrating proptosis and ophthalmoplegia of the left eye.

from a tumor behind the left eye (via inferior orbital route) was done and was reported to be a hemangioma.

Four months prior to admission to Montefiore Hospital, the patient developed weakness in the left arm and attacks of tachycardia. Three months before admission, his left leg became weak. For the last month prior to admission, it was necessary for the patient to rush to the bathroom in order to avoid soiling his clothes. For several weeks prior to admission, the patient complained of headaches.

Physical examination. The general physical examination revealed a fairly well-developed and well-nourished man. There was motor weakness of the left arm and leg. Left tendon reflexes were exaggerated. Left abdominal reflexes were absent.

Ocular examination. Visual acuity was: O.D., 15/15; O.S., finger counting nasally. Hertel exophthalmometer readings with a base line of 103 were: O.D., 20; O.S., 35 (fig. 8). The right eye was wholly normal.

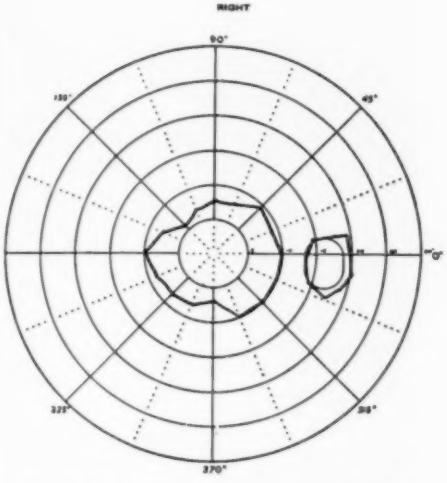

Fig. 9 (Grino and Billet). *Case 2.* Visual field for the right eye, March 10, 1948, 1/2,000 white; blindspot, 2/2,000 white. Visual acuity: 15/15.

The left eye showed restriction of the extraocular muscles in keeping with the proptosis. The left optic nervehead showed definite temporal pallor.

Visual field studies showed normal central and peripheral fields in the right eye, for 1/2,000, 1/330, and 2/330 white. The left eye retained only a small island of vision

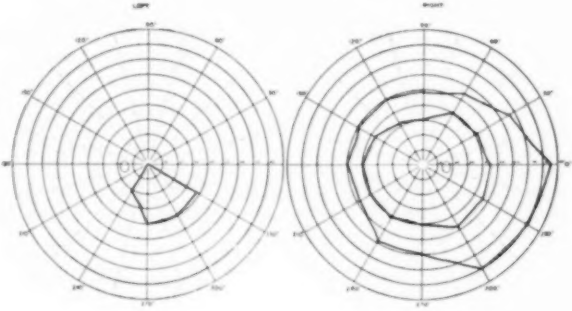

Fig. 10 (Grino and Billet). *Case 2.* Visual fields, March 10, 1948: R.E., 1/330 white; 2/330 white; L.E., 10/330 white. Visual acuity: R.E., 15/15; L.E., finger counting.

nasally and below for 10/330 white. These visual field findings suggested insult to the left optic nerve probably within the orbit and not in the cranium (figs. 9 and 10).

Electrocardiography showed a right bundle branch block without activity of any coronary lesion. Electro-encephalography showed definite electrical abnormality with concentration of disturbances anteriorly to the right of the midline. Right angiographic studies localized the cerebral tumor as a highly vascularized lesion in the right parietal region near the vertex, measuring 3 by 4 cm. Left angiography disclosed a highly vascular lesion intraorbital in location (figs. 11 and 12).

Chest X-ray films showed three oval-shaped areas of homogeneous density in the lung.

Laboratory data. Urine and blood counts were normal, as were blood urea and sugar. Wassermann was negative.

Course in hospital. On March 22, 1948, under general anesthesia, a right postero-superior frontal craniotomy for removal of the cranial lesion was performed. The brain itself bulged quite markedly through the defect in the dura mater. The lesion was disclosed to be fluorescent due to the injection of fluorescein before the operation. The tumor was removed and frozen sections revealed a hypernephroma.

Postoperatively, the patient remained drowsy. On the first postoperative day, he developed a fever of 105°F. He died on the second postoperative day. At autopsy, the left orbital contents were obtained in a body (fig. 13).

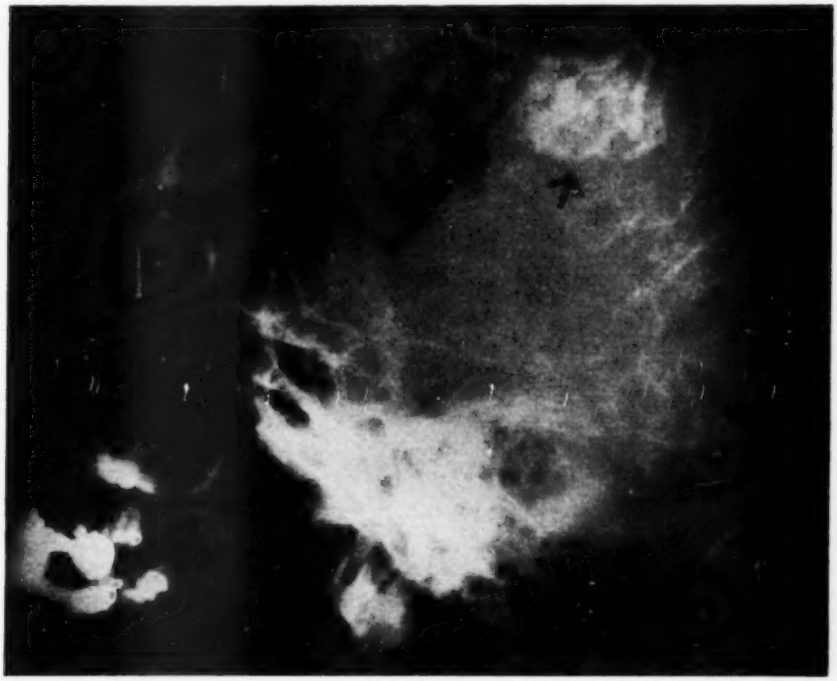

Fig. 11 (Grino and Billet). *Case 2.* Right carotid angiography, lateral view. Capillarogram showing the shadow of the brain tumor.

CASE 3

History. H. was a white woman, aged 46 years, who, in November, 1947, was told by an optometrist that he was unable to correct the vision in her right eye and that she should see an ophthalmologist. The patient had no subjective complaints but on direct questioning admitted that, when glasses were fitted in 1945, it seemed as if the lashes of the right eye brushed against the glass and that it took numerous adjustments to fit them. She, however, had noticed no prominence of the right eye. There was no history of headache, disturbances in smell, pain in the face, or endocrine disturbance. The past history was essentially noncontributory. Examination revealed a rather obese woman with a blood pressure of 160/90 mm. Hg, but no other abnormalities (fig. 14).

Laboratory data. Hemoglobin 13 gm.; R.B.C., 4.37 million; W.B.C., 10,900 (64-percent polys, 6-percent immatures, 24-percent lymphocytes, 4-percent monocytes, 2-percent eosinophils). Urinalysis was negative. B.U.N. 13.5 mg. percent; blood sugar, 82 mg. percent; total protein, 6.4 percent.

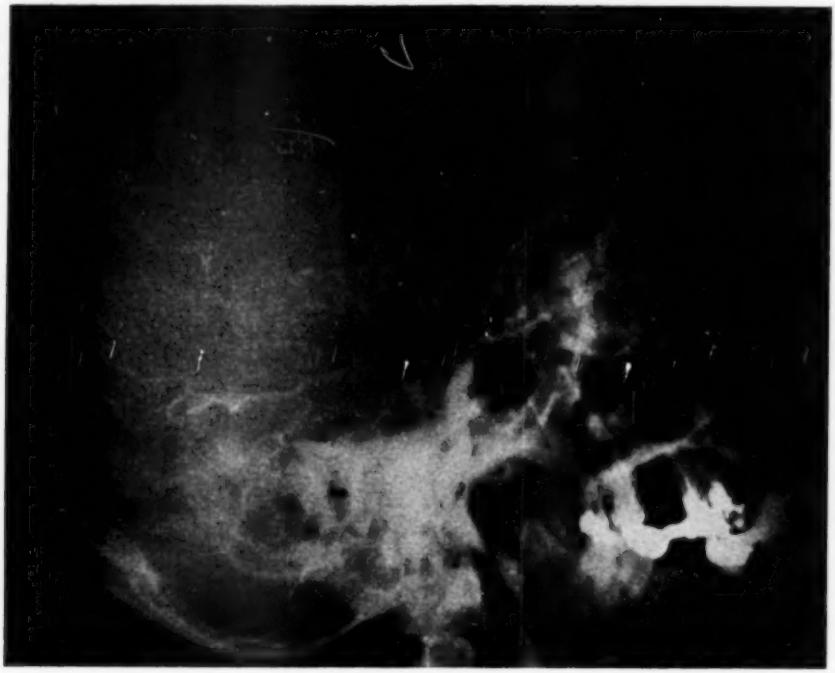

Fig. 12 (Grino and Billet). *Case 2.* Left carotid angiography, lateral view of arterial phase. The vessels of the orbital tumor are completely filled with diodrast.

Visual field studies showed normal fields for the left eye. For the right eye, there was marked peripheral contraction encroaching on the central area with marked central involvement as indicated by central field studies (figs. 18 and 19).

Eye note. Visual acuity on February 9, 1948, was: O.D., 15/30; O.S., 15/15. The right eye was more prominent than the left. Hertel exophthalmometer measurements with base-line 100: O.D., 22; O.S., 17.5. Extraocular movements were unrestricted. Fundi; O.D., showed temporal pallor and

blurring of disc margins with some fullness of veins. O.S., showed upper nasal disc margin to be blurred, slight pallor temporally, veins full.

The blindspot of the right eye could not be properly evaluated even with 20/2,000 even in the region of the optic foramen or possibly even in the orbit.

X-ray studies of the skull, including postero-anterior and lateral stereoscopic views as well as views of the optic foramina showed thickening of the right sphenoid wing and

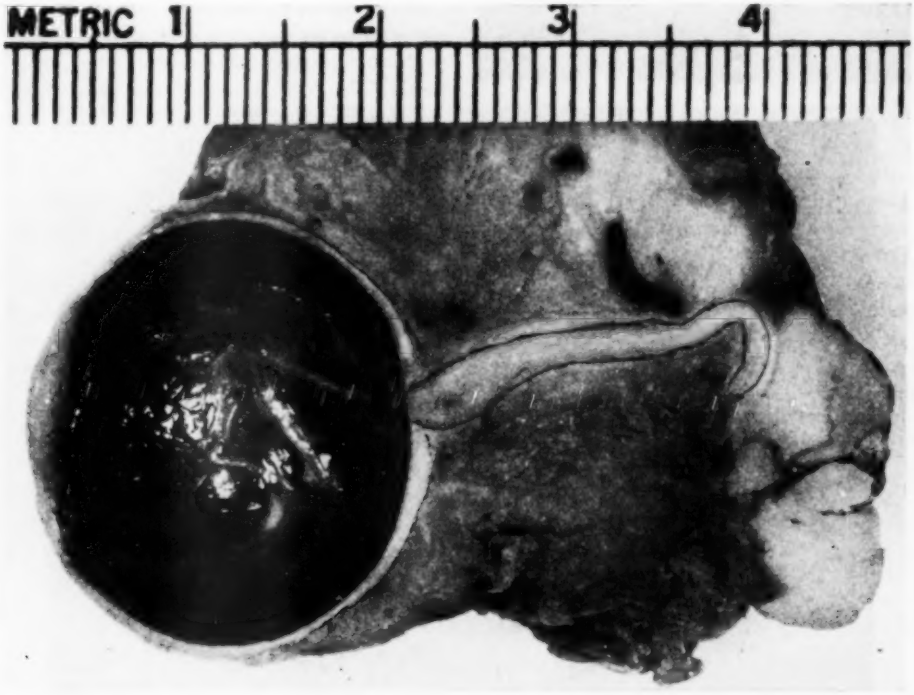

Fig. 13 (Grino and Billet). *Case 2.* Autopsy specimen. Sagittal section of the left orbit demonstrating the tumor.

white. The right eye showed practically no field for 1/2,000 white. With 15/2,000 red, there were no definite central hemianopic features in the right eye. In the left eye, the peripheral field for 2/330 white showed no substantiating criteria for upper defect in the field. The blindspot in the left eye was within normal limits.

Dr. Chamlin thought that, in view of the complete lack of involvement, one must assume that this interference with the visual pathway was limited to the right optic nerve, probably well anterior and possibly of the orbital roof on that side. There was suggestion of spicule formation in the latter area. Dr. Davidoff reported that the appearance was typical of right sphenoidal ridge meningioma (fig. 15). Electro-encephalograms showed an essentially normal electrical pattern.

Angiography performed by percutaneous injection of the carotid showed a distinct semilunar posterior concave depression of the anterior cerebral artery in the region of the bony overgrowth in the sphenoidal ridge on the right, at the medial termination of the

Fig. 14 (Grino and Billet). *Case 3.* Demonstrating slight proptosis of right eye.

sphenoidal ridge. The venogram showed the tumor by the injection of the blood vessels. The remainder of the angiogram was considered to be within normal limits (figs. 16 and 17).

Course in hospital. All investigative procedures were carried out with no untoward effects. The diagnosis of a sphenoidal ridge meningioma of the outer one third of the sphenoid ridge was fairly well established and operation was recommended. The patient at first was quite resistive to the idea of surgical intervention but eventually acquiesced and was discharged to return in about one month for surgery.

On April 15, 1948, a right frontotemporal craniotomy with orbital decompression was performed under endotracheal ether anesthesia. The bone overlying the base which was markedly thickened and vascular, was rongeured away back to the anterior clinoid process, thereby unroofing the orbit. The sphenoid ridge and part of the lesser wing of the sphenoid were removed by means of perforators, burrs, and the rongeurs.

The dura was then incised and a small hemispheric tumor, measuring about 3 to 4 cm. in diameter and ½ cm. in thickness, was encountered in the dura opposite the Sylvian vessels and removed.

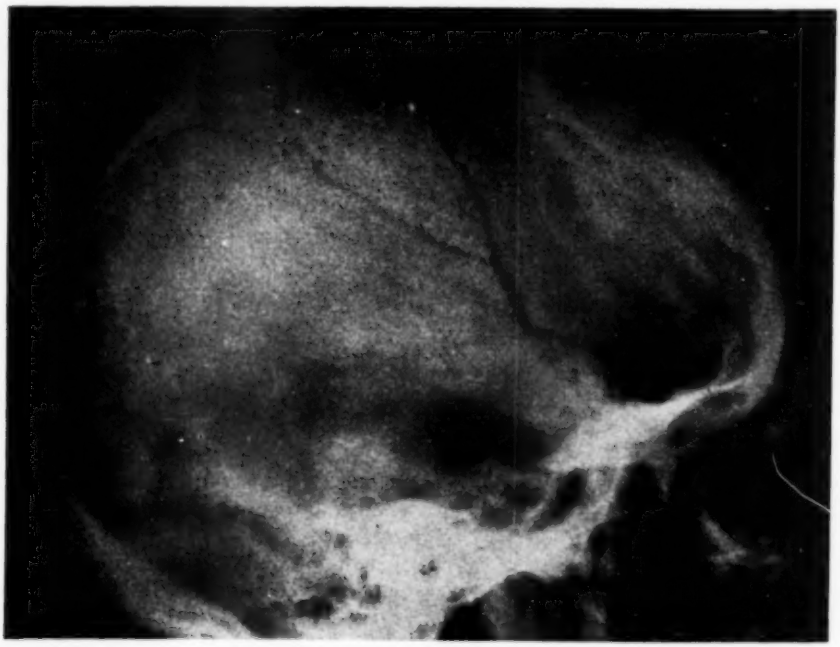

Fig. 15 (Grino and Billet). *Case 3.* Roentgenogram of the skull, right lateral view, showing some bony changes of the sphenoidal plate.

The orbital fascia was then incised and the orbital contents seemed to decompress themselves through the defect.

Postoperatively the patient did well and her wound healed by first intention. At the time of the patient's discharge, the visual acuity in the right eye was 15/15−2 as compared to 15/30 preoperatively. The exophthalmometer reading showed the same 5-mm. proptosis of the right eye.

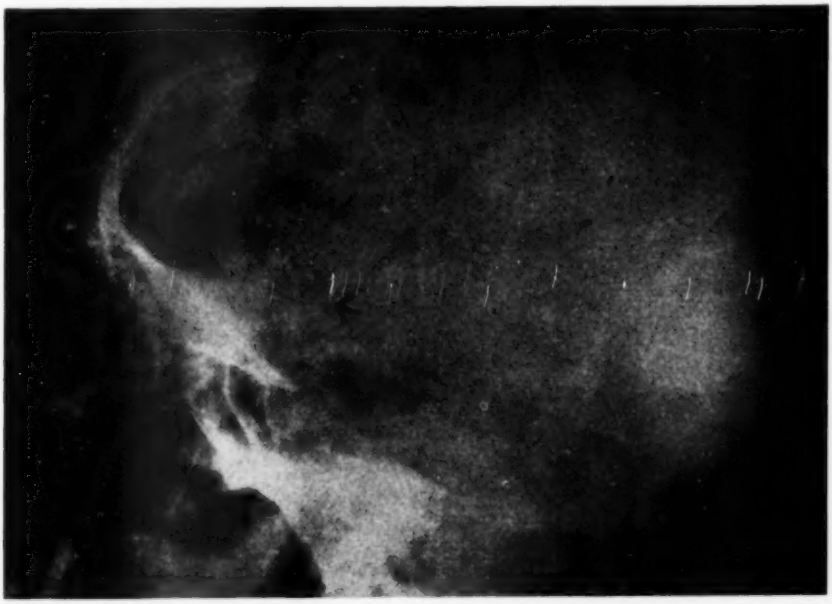

Fig. 16 (Grimo and Billet). *Case 3.* Right carotid angiography, lateral view. Capillarogram showing vascular network of tumor filled with diodrast.

Pathologic report. The tumor was a meningocytic meningioma.

Discussion

The three cases here presented, two orbital tumors (one primary, the other metastatic) and an intracranial tumor located at the sphenoidal wing, illustrate the difficulties in making the correct differential diagnosis in cases of unilateral exophthalmos. In spite of their different natures and locations, their histories, as well as their clinical and roentgenologic findings, show a marked degree of similarity. Unilateral exophthalmos and gradual loss of vision were the most prominent findings of the three, while roentgenologic changes also were present in greater or less degree in Cases 1 and 2.

That angiography has been not only useful but indispensable in making these diagnoses is evidenced by the accompanying angiograms, in which the tumors are clearly demonstrated, as well as by a close examination of the case histories (figs. 6, 11, 12, 16, and 17).

In Case 1, given the findings obtained by the various examinations—unilateral exophthalmos, partial loss of vision, bony changes in the sphenoidal wing of the affected side, and the aspects of the visual fields —one would suspect an intracranial lesion, which had to be ruled out. To this end, lateral and postero-anterior stereoscopic X rays of the skull were taken, as well as X rays of the optic foramina.

Although the latter were shown to be

normal, a suspicious increase in density was found at the level of the superior orbital plate and at the greater wing of the sphenoid of the affected side. An air injection behind the globe of the eye did not yield any data of diagnostic value, resulting only in marked ecchymosis of the eyelids, which caused the patient considerable pain and discomfort (fig. 5).

It is evident in this case that, because of inability to demonstrate an orbital tumor by the means at our disposal (angiography excepted), one would decide upon an intracranial exploration (as was done in Case 2) or upon temporizing measures which could only result in further loss of vision.

Cerebral angiography was finally performed by the percutaneous method which showed unmistakably, in all three phases, the presence of an orbital tumor. This is one of the few cases in our series of angiographies in which general anesthesia was employed because, at that time, we were developing the percutaneous technique and we needed the maximum relaxation of the patient.

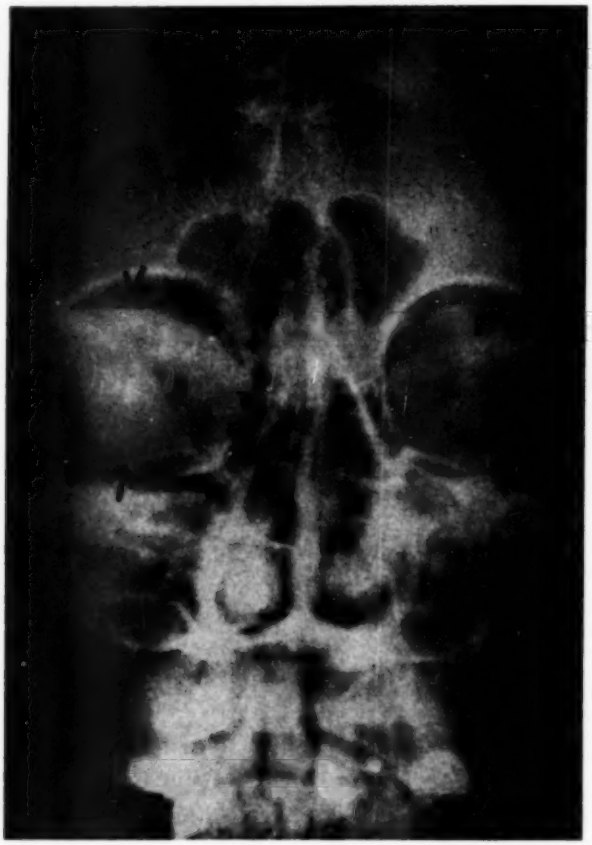

Fig. 17 (Grino and Billet). *Case 3.* Right carotid angiography, antero-posterior view, showing vascular bed of tumor filled with diodrast. The arrows point to the tumor.

As can be seen by the angiogram (fig. 6), this tumor is eminently vascular. Although we were interested in determining its nature, we did not think that biopsy would be a safe procedure, especially in view of the untoward results of the air injection. Considering further the reluctance of the patient to accept more radical measures and the fact that a great percentage of orbital tumors are of hemangiomatous type (Reese[7]), we agreed upon X-ray therapy and periodic examinations of the visual fields, anticipating, however, that we should have to insist upon surgical treatment, should the condition become worse.

A point of interest in this case is the relation of the ophthalmic findings to those of the angiographic. The visual fields, which suggest (in the words of Dr. Chamlin) "interference with the right optic nerve retrobulbarly, extending rather far back toward the chiasmal area," can be easily correlated to the angiogram which shows a small number of abnormal vessels above the lesser wing of the sphenoid on the affected side.

When Case 2 was first seen by the neurosurgeon, the history (except for the nephrectomy) and the findings were almost identical to those described in the preceding case. Because a pathologic report could not be obtained, the reasons for the nephrectomy were never established.

Although neither an intraorbital, nor an intracranial tumor could be ruled out on the basis of the clinical and radiologic findings alone, nevertheless an intracranial exploration was undoubtedly indicated, if only for the purpose of decompressing the optic nerve in the event that no tumor was found. This treatment would not change essentially, however strongly one might suspect the presence

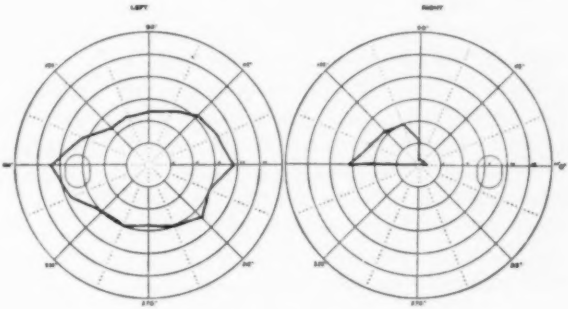

Fig. 18 (Grino and Billet). *Case 3.* Visual fields on February 9, 1948: R.E., 2/2,000 white; L.E., 1/2,000 white. Visual acuity: R.E., 15/30; L.E., 15/15.

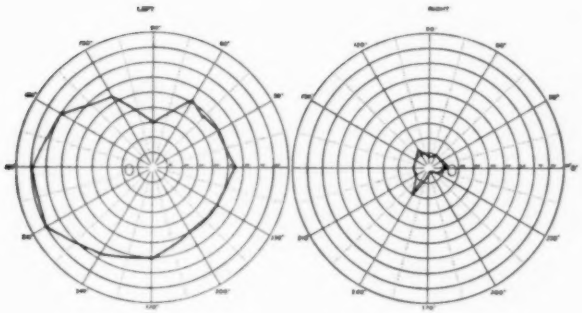

Fig. 19 (Grino and Billet). *Case 3.* Visual fields on February 9, 1948: R.E., 2/330 white; L.E., 2/330 white. Visual acuity: R.E., 15/30; L.E., 15/15.

of a metastasis, since we know that two different types of tumor can be present in the same individual, as has been emphasized lately by Feiring and Davidoff.[3]

Three years after the intracranial exploration, which was reported to be negative, the ocular signs were so obvious that the problem was not one of localization but of knowing the nature of the tumor. A biopsy was then performed, the report from which was "possible hemangioma."

Seven months later the patient developed a weakness on the left side of his body which was the immediate cause of his admission to Montefiore Hospital, where angiography of the left carotid system was performed in order to rule out any possible intracranial extension of the orbital tumor (fig. 12). Since this patient had neurologic signs which could be related to a possible lesion in the right cerebral hemisphere, the right carotid artery was also injected percutaneously two days afterward and a tumor was shown situated in the right frontal lobe (fig. 11).

In resume, this patient had, from the beginning, a metastatic lesion in his left orbit which was impossible to discover in its early stages and which probably could have been revealed by angiography, had angiography been available. As previously mentioned, the tumor mass removed at operation, as well as the orbital tumor obtained at necropsy, was shown to be a hypernephroma.

Case 3 had a history and presented clinical findings similar to those of the preceding cases. This patient had a unilateral exophthalmos with gradual loss of vision. Her radiologic findings, however, were compatible with an intracranial growth, showing bony changes commonly present in sphenoidal-wing meningiomas. Although the preoperative diagnosis was confirmed in the operation and was finally substantiated by the histologic report, it is evident that many doubts could have been cast upon that diagnosis without the documentary proof given by angiography (figs. 16 and 17).

More important still, from the point of view of the surgeon, was the fact that no intraorbital extension could be demonstrated angiographically. The steady improvement of the patient after the operation, in which the orbit was unroofed but not explored, testifies that no orbital tumor was present and confirms the assumption derived from the angiographic data.

At this point, we should like to mention that the absence of a tumor shadow in the angiogram does not preclude the existence of a tumor. Not all tumors have a well-developed vascular supply; astrocytomas and melanomas, for instance, are poorly vascularized and very rarely will be demonstrated by angiography. However, in these and allied cases, angiographic diagnosis is still made possible by the displacement of the vessels, as this is as much indicative of a growth as is a tumor's shadow.

However far from the scope of this paper it may seem to be, we wish to make some remarks upon the treatment of these cases, which will help to justify the emphasis that we place upon the differential diagnosis.

A complete removal of the tumor, here as elsewhere in the body, is the surgeon's aim. The removal of an intraorbital tumor or an intracranial tumor with orbital extension is, however, a major operation and not, by any means, an easy one, requiring perfect familiarity with the region, as well as knowledge of the suspected pathologic condition.

It is evident that, if the exact situation of the tumor is known and some hint of its pathology is obtained, the surgeon will be enabled to explore the intraorbital cavity with the least possible damage to the important structures that it contains.

That the removal of an intraorbital tumor by an intracranial approach is not only feasible but the route of choice in the majority of cases has been demonstrated by Cushing[1] and Dandy.[2] This approach is employed routinely by neurosurgeons. The operation consists of a frontal craniotomy, as for the

exploration of the pituitary gland, followed by the removal of the orbital roof. The capsule of the orbit is then opened widely, which allows a thorough exploration of the orbital cavity.

It is evident that one would feel reluctant to explore the orbital cavity unless the presence of a tumor were well demonstrated in advance, since the danger of damaging important structures cannot be overemphasized. Since one cannot, however, neglect such a tumor, it is of the utmost importance to have an accurate diagnosis. Although our experience is not extensive, we feel that angiography is the safest and most helpful method for differential diagnosis in cases which may show either intraorbital or intracranial tumors.

Summary and Conclusions

Two cases of orbital tumor and one of intracranial tumor are presented.

In all three cases the history and symptoms were similar.

Accurate localization was made possible by angiographic visualization of the tumors.

The importance of accurate localization as a prerequisite for proper surgical treatment is emphasized.

Cerebral angiography, by either the direct (percutaneous) or the open method, is presented as a harmless procedure, permitting radiologic visualization of orbital, as well as intracranial, tumors.

The technique of cerebral angiography is outlined.

We feel that cerebral angiography should be used in the diagnosis of cases exhibiting unilateral exophthalmos, in preference to more dangerous or less informative methods, as, for instance, pneumoencephalography, puncture biopsy, and intraorbital air injection.

71 East 77th Street (21).

The authors are indebted to Dr. Leo M. Davidoff for the liberal use of his cases as well as for his constant encouragement, and to Dr. Samuel Gartner whose suggestion initiated the writing of the paper and who was kind enough to read the manuscript. The cooperation of Dr. Max Chamlin in interpreting the visual fields has been invaluable. We wish to thank Dr. Raymond E. Weston for suggesting to us the use of the Cournand needle.

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A SURGICAL TREATMENT FOR PTERYGIUM BASED ON NEW CONCEPTS AS TO ITS NATURE*

H. Saul Sugar, M.D.
Detroit, Michigan

Although much has been written regarding the cause of pterygium, the frequency of recurrence after surgical treatment is indicative of our profound ignorance of its true nature. As a result of clinical and histologic observations begun in 1944 on military personnel, and continued since, a theory of the nature of pterygium was formulated and a new method of surgical treatment devised. The results in approximately 50 cases have been such as to warrant this communication, even though follow-up in the early military cases was not possible.

According to Kamel,[1] "a true pterygium is a pathological encroachment of part of the bulbar conjunctiva exposed in the palpebral tissue over the cornea. This encroachment is in the form of a fold of the conjunctiva, triangular in shape, with the apex, called the head, on the cornea, the base merging imperceptibly in the caruncle region or the region of the outer canthus, and the sides formed of two folds of the conjunctiva, an upper and a lower." It is considered by Duke-Elder[2] to be a degenerative process which essentially affects the cornea.

These authors, like nearly all other writers on the subject, are concerned with the cornea and conjunctiva. I believe the layer of Tenon's capsule lying in the interpalpebral zone between the conjunctiva and sclera of cornea to be the essential element in the occurrence, extension, and recurrence of true pterygium.

Clinical observations

The first clinical observations were made on eyes with pingueculae and early pterygia to determine the relationship between the two, and the earliest onset of pterygium. It was noted that all early pterygia were associated with pingueculae and that in each instance the pinguecula was elevated sufficiently to raise the epithelium mechanically from the end of Bowman's membrane. This suggested that the latter is the barrier separating the pinguecula from corneal encroachment when it is then termed pterygium. Once the barrier had been passed, the conjunctiva appeared to be pulled onto the cornea, as shown by the fact that the pinguecula becomes the apex of the pterygium (fig. 1) and by the folding of the conjunctiva which, when released surgically, returns to the normal conjunctival position.

The view that the pterygium develops from a pinguecula was first introduced by Zehender,[3] in 1869, although Horner[4] may deserve credit for originating this theory. Fuchs[5] championed this theory and, although Hubner[6] stated that pterygium could occur independently, it is difficult to be certain of this once a pterygium of sufficient size has formed, since the pinguecula is carried onto the cornea as the head of the pterygium and may leave no evidence of its previous presence in its usual position.

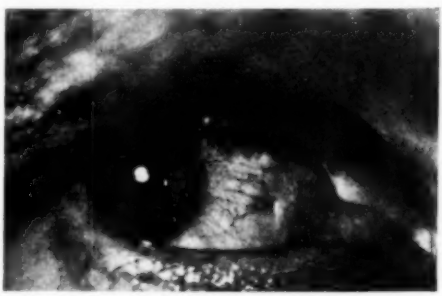

Fig. 1 (Sugar). Pterygium showing vascularity and pinguecula forming its head.

* From the Wayne University Medical School and Receiving Hospital. This work was largely done at the Barnes General Hospital (U.S. Army).

Observations made during surgery

Further observations made during surgical procedures on pterygiums showed that under the conjunctiva there is a definite layer of vascularized tissue (Tenon's capsule) which, in the horizontal meridian, has the appearance of a thin muscle tendon. It continues to be the very head of the pterygium.

Histologically, this tissue contains hypertrophic and hyperplastic elastic fibers which lie parallel to each other, thus giving rise to the "tendinous" appearance. This layer separates easily from the conjunctiva and is practically free from the sclera.

In addition, some of the prominent horizontal blood vessels extending from the inner canthal region toward the head of the pterygium lie in this tissue and not in the conjunctiva itself (fig. 1). It appears that these are anterior ciliary vessels, which lie just anterior to the rectus muscles in Tenon's capsule, which are pulled across the sclera and cornea with Tenon's capsule.

Just anterior to the head of the pterygium, there is a halo of grayish-white opacity, a short distance in front of which are islands of superficial opacities that, histologically, have been found to be small vesiclelike formations in Bowman's membrane at the points where the corneal nerves pierce the latter. Fuchs[5] has described these changes well.

When the head of the pterygium is removed, it is noted that there are dense adhesions between the pterygium tissue and cornea in some parts, while in other parts the two separate easily.

Histologic observations

Aside from clinical observations, there are important histologic observations which are well known and important in formulating the theory of the nature of pterygium. Most important is the histologic similarity between the pinguecula and the body of a pterygium. Both are covered by conjunctival epithelium with variations which are not essential to the process of extension of the pterygium.

Fig. 2 (Sugar). Photomicrograph showing the dense layer of Tenon's capsule (A) and calcified areas at (B). The thin strands which lie under Tenon's capsule are seen at (C).

Near the surface of its head and neck, the pterygium is covered by flat cells, with cylindrical cells in the folds and furrows and at the base. Bowman's membrane is destroyed in places and here the superficial corneal lamellae are involved in the pterygium.

The stroma of each shows a moderately vascular areolar tissue structure, loose in the early stages and more compact later (fig. 2). There is hyaline degeneration of fibrous tissue and deposition of amorphous hyaline and sometimes chalk deposits. Hypertrophy and hyperplasia of the elastic fibers are quite prominent.

Discussion

These observations have led to the following incomplete theory: A pterygium develops from a pinguecula. The latter is a degenerative change in the subconjunctival fascia (Tenon's fascia). Two factors may be significant in this degeneration.

1. The closure movements of the lids squeezing the conjunctiva between them.

2. The movements of the horizontal rectus may cause intermittent pull on Tenon's fascia. That this factor alone could not be effective is shown by the absence of these changes in the region of the vertical recti.

The degenerative process leads to hyperplasia and hypertrophy of the elastic tissue and deposition of hyaline which causes an elevation that eventually separates the epithelium from Bowman's membrane. Then, probably because of a chemotactic force either in the nature of the changes occurring when tendinous tissue attaches itself to bone or other tissue, perhaps as a response to the pull of the "tendinous" Tenon's fascia, or as a response to a nutritional demand, changes occur in the cornea adjacent to the head of the pterygium and lead to the formation of connective tissue which contracts, slowly pulling the fascial layer onto the cornea. As the process repeats itself the tissue encroaches further onto the cornea, pulling the conjunctiva with it.

This theory explains the recurrences produced by all methods of pterygium surgery in which the subconjunctival tissue is permitted to remain.

This theory considers pterygium to be a purely degenerative process. On the other hand, Kamel believed the process to be inflammatory and D'Ombain[7] held that it is an irritative disease due to exposure and not primarily a degeneration, but he stressed that it is secondarily a degeneration. Kamel felt that a keratoconjunctivitis occurs, with the laying down of fibrous bands which contract and cause encroachment of conjunctiva onto the cornea. He treated pterygium by separating the conjunctiva from the underlying layer and cauterizing subconjunctivally with phenol on a toothpick.

New surgical procedure

On the basis of the theory herein presented, the following surgical procedure has been used successfully. No recurrence has been seen. None is expected, since the operation is based on the formation of a sclero-conjunctival adhesion, 4-mm. wide adjacent to the limbus.

Anesthesia is induced by the use of 2 or 3 installations of 4-percent cocaine-hydrochloride solution at 2-minute intervals. Then, a small amount of 2-percent procaine solution, enough to raise a thin bleb between the sclera and Tenon's capsule, is introduced through a hypodermic needle beneath the body of the pterygium about 4 to 5 mm. from the limbus.

Incision. After about five minutes, an incision (fig. 3) is made in the conjunctiva just posterior to the head of the pterygium, extending all the way from its lower to its upper border and then laterally at each border to the limbus.

This incision separates the conjunctiva from the cornea and permits tthe entire conjunctiva, forming the surface of the pterygium, to be undermined to the plica semilunaris, and to a couple of millimeters beyond its upper and lower borders. The conjunctiva is thus freed from attachment to the subconjunctival layer and may resume its normal position.

A second incision is now made at the same location as the first, this one being made through the "tendinous" layer of the Tenon's capsule tissue, from the lower to the upper border of the pterygium at this point.

A scissors is inserted between this layer and the cornea and sclera, and this layer is undermined for about 4.5 to 5.5 mm. from the limbus, and extending about a millimeter beyond the upper and lower borders of the pterygium. The portion of Tenon's capsule, which is thus undermined, is excised.

The head of the pterygium is now shaved from the cornea in such a manner as to remove as much of the scar tissue as possible, leaving the limbus free.

Suture. A double-armed silk suture is inserted through the superficial layers of the sclera about 3.5 mm. from the limbus and parallel to the latter, and each end is passed through the conjunctiva near its cut edge. The suture is tied over the conjunctiva, leaving a bare area of 3 mm. between the cornea and conjunctiva.

A patch is applied. Ointments are used after the first day. The suture is removed on the fourth day. The suture may be dis-

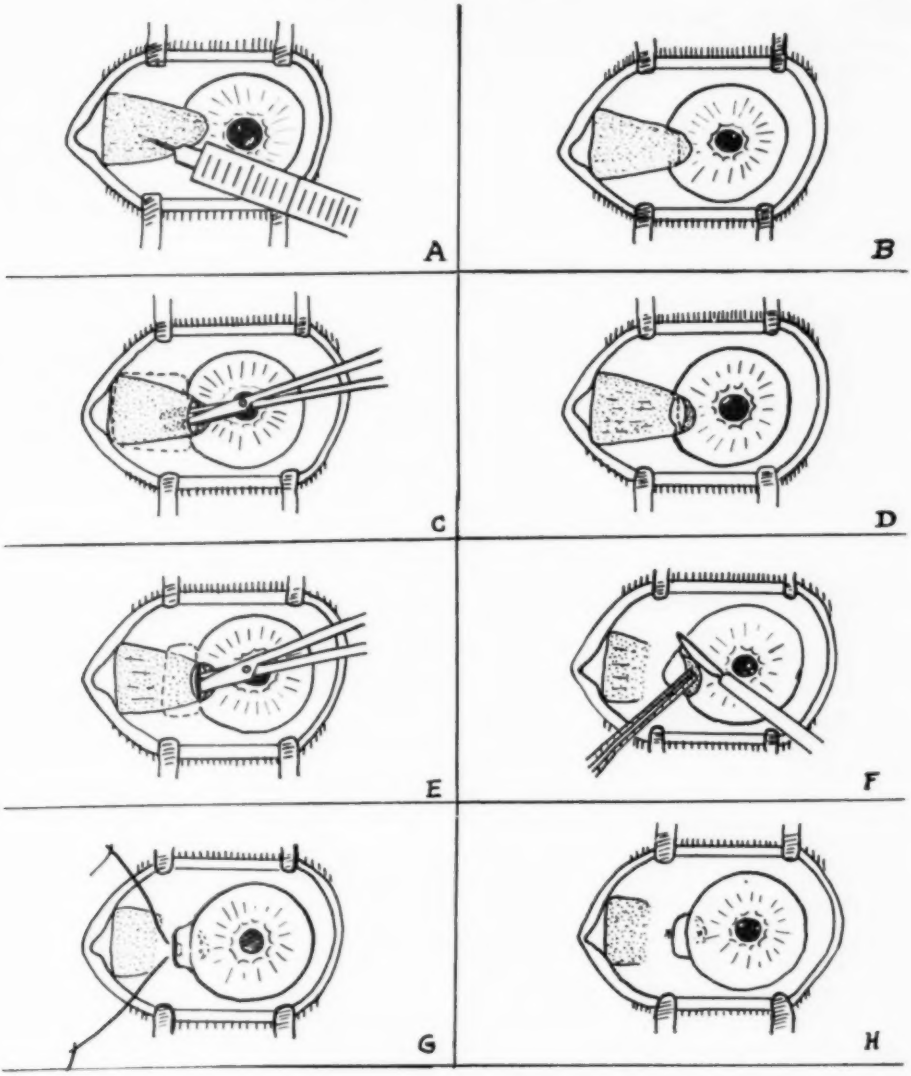

Fig. 3 (Sugar). *Steps in the operative procedure.* (A) Site of procaine injection for anesthesia. (B) Site of conjunctival incision. (C) Undermining of the conjunctiva. Broken lines indicate limits of undermining. (D) Site of incision in subconjunctival "tendinous" layer. (E) Broken lines indicate limits of area of subconjunctival layer undermined and excised. (F) Shaving of head of pterygium from cornea. (G) Insertion of suture (scleroconjunctival). (H) Suture tied leaving 3 mm. of bare sclera.

pensed with if the surgeon desires. This tends to lessen the tissue reaction considerably.

Comment. This procedure is simple enough to be done in the office, if adequate facilities for sterility are available. It is surgically sound as compared to the McReynold's operation, in which epithelium is

buried. The eye becomes pale much more quickly than with any operation in which the subconjunctival tissue is permitted to remain. The formation of the adhesion between the conjunctiva and sclera and the epithelization of the bare area are rapid.

In some cases, an adhesion of conjunctiva to the area of corneal scar, where the head of the pterygium had been, gives the impression of recurrence, but this has not been observed to continue further onto the cornea.

There has recently appeared another pterygium operation based on similar principles (D'Ombain[7]). In fact, it has been the stimulus of D'Ombain's paper which has led to the actual writing of this one.

In his procedure, the entire subconjunctival tissue is removed between the cornea and the plica semilunaris and a 5-mm., bare area of sclera left to epithelize the unsutured conjunctival edge. This procedure should lead to good results but has the disadvantage of possible injury to the horizontal rectus muscles while removing the Tenon's tissue since the internus inserts only 5.5 mm. from the limbus.

Conclusion

A theory is presented that the development of pterygium from pinguecula is a degenerative process in which the hypertrophic and degenerative changes in Tenon's capsule in the interpalpebral zone are considered to play the essential part. A simple, effective operative procedure based on this theory is presented.

311 Kales Building (26).

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Historical Miniature

Egyptian Ophthalmology

To "clear the pupil" a solution of saltpeter and ebony was used. The nature of this ailment is difficult to determine. The hieroglyphic sign for it was the crocodile (= the terrible). It may have been cataract.

Hirschberg, *Graefe-Saemisch Handbuch.*

RETROLENTAL FIBROPLASIA IN PREMATURELY BORN CHILDREN*

ANITA PEEK GILGER, M.D.
Cleveland, Ohio

The purpose of this study was twofold. The first was to determine the incidence of retrolental fibroplasia in prematurely born children, together with any factors in the development of the disease which such a survey might unearth. The second purpose was to attempt to gain a better knowledge of the clinical course of this condition by observing a group of prematurely born infants at regular intervals from birth.

Terry[1] stated that there was an apparent variation in the frequency of retrolental fibroplasia in different medical centers. It was, therefore, decided to conduct a survey in a middle-western community. In 1945, Terry[2] stated that Clifford had found in a series of less than 50 infants at the Boston Lying-In Hospital an incidence of retrolental fibroplasia of 12 percent in infants weighing 1,307 gm. or less at birth. In 1948, Clifford and Weller[3] quoted Allers as saying that 23 percent of the infants weighing between 2 to 3 pounds at birth, at the same hospital, develop retrolental fibroplasia. William and Ella Owens[4] found, in premature infants born between 1945 and 1947 at The Johns Hopkins Hospital, 5 cases of retrolental fibroplasia in 83 observed cases with birth weights of 1,899 gm. or less, an incidence of 6 percent.

Reese[5] states that there has been an apparent increase in retrolental fibroplasia in the last few years. This may possibly be due to improved survival of the babies whose birth weights were 1,814 gm. or less. He also states that 40 to 60 percent of the mothers of his patients with retrolental fibroplasia gave a history of uterine bleeding during pregnancy.

Vitamin A as a possible etiologic factor has been considered since Warkany[6] found a fibrous retrolenticular membrane in place of the vitreous in rats with maternal vitamin-A deficiency. Clifford and Weller[3] state that in their series of cases, the postnatal administration of absorbable, water-soluble vitamin A did not prevent the later development of retrolental fibroplasia.

Bakwin[7] suggested the possibility that retrolental fibroplasia was sex-linked because, out of 150 patients reported by Reese and Terry, 95 were males. Krause[8] described retrolental fibroplasia as a part of a congenital encephalo-ophthalmic dysplasia of unknown origin. He stated that, if the child was examined after the age of four years, the ocular disease rarely occurred without clinical signs of involvement of the brain.

METHOD

This survey included 229 children with a birth weight of less than 2,268 gm. (5 pounds) who were either born at, or admitted before the age of 24 hours to, the Cincinnati General Hospital during the 5-year period from January, 1943, to January, 1948. None of the infants born during this period was a private patient. Form letters or appointments on hospital discharge, seconded by home visits of public health nurses, were the methods of inducing the parents to bring the children to the clinic.

For a 6-month period, from July, 1947, to January, 1948, every infant born in the hospital weighing less than 2,268 gm. was examined within the first week of life and every week to two weeks thereafter during the hospital stay. After discharge, the babies of this group whose parents coöperated were examined at monthly intervals until the baby became at least six months of age.

In addition to this group, children born between January, 1943, and July, 1947, were

* From the Ophthalmology Department and the Pediatrics Department of the University of Cincinnati Medical School, Cincinnati, Ohio.

examined; but only one clinic visit was made and that at a time when the child had attained the age of one year or more.

In every case, examination consisted of inspection of the external segment of the eyes and ophthalmoscopic examination following pupillary dilatation with either homatropine hydrobromide or neosynephrin hydrochloride (ophthalmic). Other examinations were performed as indicated. With very rare exceptions, the only sedation required for satisfactory examination of the infants was either formula or glucose water.

Incidence

In the 5-year period, 1943 to 1948, there were 507 babies born at the Cincinnati General Hospital weighing less than 2,268 gm. at birth and presumably living at the time of this study (table 1). Of this total, 156 weighed 1,814 gm. (4 pounds) or less. Of the total group, 229, or 45.2 percent, were examined; 96, or 61.5 percent of those with birth weights of 1,814 gm. or less were seen.

This higher incidence of returns was probably not due to the greater concern of the mothers of the smaller babies but to the

TABLE 1

Incidence of retrolental fibroplasia in premature children

Year	Birth Weight (gm.)	Total Living	Lost or Moved		Infants Seen		Retrolental Fibroplasia	
			No.	%	No.	%	No.	%
1943	1,361 or less	1	0	25.0	0	0.0	0	
	1,362–1,814	11	3		3	27.3	0	
	1,815–2,000	33	7		6	18.2	0	
	2,001–2,268	51	8		16	31.4	0	
	Total	96	18	18.8	25	26.1	0	
1944	1,361 or less	4	1	47.3	3	75.0	0	
	1,362–1,814	15	8		6	40.0	0	
	1,815–2,000	13	3		6	46.2	0	
	2,001–2,268	23	9		8	34.8	0	
	Total	55	21	38.2	23	41.8	0	
1945	1,361 or less	4	1	18.5	3	75.0	0	7.1
	1,362–1,814	23	4		11	47.8	1	
	1,815–2,000	14	4		4	28.6	0	
	2,001–2,268	45	18		16	35.6	0	
	Total	86	27	31.4	34	39.5	1	2.9
1946	1,361 or less	14	1	12.0	12	85.7	1	5.6
	1,362–1,814	36	5		24	66.7	1	
	1,815–2,000	28	9		9	32.2	0	
	2,001–2,268	35	8		16	45.7	0	
	Total	114	23	20.2	61	53.5	2	3.3
1947	1,361 or less	6	2	17.0	4	66.7	1	11.8
	1,362–1,814	41	6		30	73.2	3	
	1,815–2,000	30	5		14	46.7	0	
	2,001–2,268	80	10		38	47.5	0	
	Total	157	23	14.7	85	54.2	4	4.7
Total 1943 to 1948	1,361 or less	31	5	19.9	22	70.9	2	7.3
	1,362–1,814	125	26		74	59.2	5	
	1,815–2,000	118	28		39	33.1	0	
	2,001–2,268	233	53		94	40.3	0	
	Total	507	112	22.1	229	45.2	7	3.1

fact that in the lower-weight group repeated nurse visits or telephone calls were made when appointments were broken, a follow-up which time and personnel did not permit for the higher weight group. Of the total group, 112 or 22.1 percent had moved away from the Cincinnati or the post-office area and nurses had lost their addresses at the time of the study. The remaining 32.7 percent could not be persuaded to come to the clinic.

Retrolental fibroplasia occurred in 7 patients out of the total of 229 seen, an incidence of 3.1 percent. However, the disease was not seen in any children whose birth weights were above 1,814 gm. Its incidence in the 96 with birth weights of 1,814 gm. or less, who were examined, was 7.3 percent.

These percentages of incidence of retrolental fibroplasia are probably higher than they would have been if it had been possible to examine all the children available in the Cincinnati area. Of the children with birth weights of 1,814 gm. or less, there were 29 unobserved children, or 18.6 percent who were living within the Cincinnati area at known addresses at the time of the study and whose mothers received a minimum of two appointments which were broken.

These mothers had been visited by public health nurses who usually saw the children and reported no obvious gross defects of the eyes. Although it is impossible to exclude the existence of retrolental fibroplasia merely by external examination, it is unlikely that any of the 29 unexamined children had such an extensive retrolental membrane that the undilated pupillary area appeared white. If this hypothesis were taken to be true, the incidence of retrolental fibroplasia in the babies weighing 1,814 gm. or less at birth would be close to 7 out of 125, or 5.6 percent.

Of the 7 cases of retrolental fibroplasia, 2 had birth weights of less than 1,361 gm. (3 pounds) and 5 had birth weights between 1,362 and 1,814 gm. (table 2). In this study the data failed to reveal any significant difference in incidence between these two weight groups.*

TABLE 2

WEIGHT DISTRIBUTION OF OBSERVED PREMATURES

Birth Weight (gm.)	Cases Seen	Cases of Retrolental Fibroplasia
700– 799	1	0
800– 899	1	1
900– 999	2	0
1,000–1,099	2	1
1,100–1,199	5	0
1,200–1,299	7	0
1,300–1,399	8	1
1,400–1,499	10	0
1,500–1,599	13	0
1,600–1,699	17	0
1,700–1,799	24	3
1,800–1,899	24	1
1,900–1,999	18	0
2,000–2,099	29	0
2,100–2,199	41	0
2,200–2,268	27	0
Total	229	

Although it may seem from Table 1 that the increase in incidence of retrolental fibroplasia from 0 to 11.8 percent in the lower weight group during the 5-year period of this study is significant, this is not the case. When a division of the examined prematures with birth weights of 1,814 gm. or less into later and earlier cases with respect to birth dates is made, the earlier half, or 48, had one case of retrolental fibroplasia; whereas, the latter half had 6 cases ($X^2 = 2.459$; $P = > 0.10 < 0.20$).

ETIOLOGIC FACTORS

In considering possible pre- and postnatal events which may affect the development of retrolental fibroplasia, only the smaller prematures (1,814 gm. or less) were investigated. This was done not only because of the fact that it was only in this group that the disease developed, but also because the heavier weight group was usually discharged from the hospital before the age of one week so that statistics concerning postnatal management and care would be dependent upon maternal memory.

* Statistical significance has been determined in this study by the method of Chi square, using four-cell contingency tables wherever possible. Yate's correction was used in all four-cell contingency tables with small frequencies.

Survival rates

The investigation of a possible increased survival rate in recent years of the smaller premature infants falls into two groups: (1) Survival of prematurely born infants to the time of discharge from the hospital, and (2) survival after hospital discharge to the age of six months or one year, by which time the existence of retrolental fibroplasia could be determined.

The rate of survival to discharge from the hospital for the 5-year period, 1943 to 1948, is shown in Table 3. Only liveborn premature infants are included, evidence of life being heart-beating or breathing. The percentage of living babies weighing 1,814 gm. or less increased from 29.1 percent in 1943 to 51.7 percent in 1947. This is not statistically significant but is suggestive that further analysis of the figures might show some significance ($X^2 = 12.310$; for $n = 4$ $P = > 0.01 < 0.02$). When the birth weights are broken down into three groups, it is found that the group of children with birth weights between 1,362 and 1,814 gm.* does have a statistically significant increase of survivals to hospital discharge ($X^2 = 15.447$; for $n = 4$, $P = < 0.01$).

In Table 4, the known survival rate from hospital discharge to one year of age is shown. Those cases listed as surviving have been seen either by me or a public health nurse, or in a Babies' Milk Fund Association Clinic after the age of either six months or one year had been attained. Out of 162 discharges, 22, or 13.6 percent of the children either were lost or had moved out of the Cincinnati area before the age of one year. Only 6 or 4.3 percent of the children not lost are known to have died during the first year of life.

TABLE 3

Survival to hospital discharge of prematures with birth weights of 1,814 grams or less

Year Born	Birth Weight (gm.)	Infants Born Living	Infants Surviving to Hospital Discharge	% Surviving to Hospital Discharge
1943	907 or less	5	0	0
	908–1,361	16	1	6.2
	1,362–1,814	34	15	44.1
	Total	55	16	29.1
1944	907 or less	5	1	20.0
	908–1,361	13	3	23.1
	1,362–1,814	40	15	37.5
	Total	58	19	32.8
1945	907 or less	5	0	0
	908–1,361	16	5	31.2
	1,362–1,814	38	25	65.8
	Total	59	30	50.8
1946	907 or less	16	1	6.2
	908–1,361	30	13	43.3
	1,362–1,814	54	35	64.8
	Total	100	49	49.0
1947	907 or less	10	1	10.0
	908–1,361	23	5	21.7
	1,362–1,814	60	42	70.0
	Total	93	48	51.7

Prenatal influences

Because the observed infants who later

developed retrolental fibroplasia had normal eyes when seen in the first week of life, it has been said that the causative factor or factors would be more likely to occur during postnatal rather than prenatal life. However, the maternal histories of prematures with birth weights of 1,814 grams or less were studied for possible clues.

In Table 5, the history of bleeding prior to onset of labor is given for mothers of observed smaller prematures in the 5-year period of the study. Bleeding varied in degree from spotting to an amount requiring hospitalization and transfusion. Slight bleeding occurring at the onset of labor was not considered abnormal. The one case of retrolental fibroplasia with maternal bleeding had only two episodes of staining. In the observed group not having retrolental fibroplasia, 19.8 percent of the mothers gave a history of bleeding during pregnancy, while in the group with retrolental fibroplasia, the figure was 16.7 percent. The difference in incidence of bleeding between the two groups is not statistically significant.

The following conditions which may have something to do with the production of normal eyes or eyes with retrolental fibroplasia were studied, but the figures failed to reveal any statistically significant differences.

1. Age at delivery
2. Parity
3. Occurrence of virus infections during pregnancy
4. Occurrence of chronic illness during pregnancy (cardiovascular disease, diabetes, tuberculosis, syphilis)
5. Cause of premature onset of labor
6. Race

When the known causes of premature onset of labor are separated, the probability that the difference between normal eyes and those with retrolental fibroplasia is due to any factor other than chance becomes even less than when the causes are grouped into known and idiopathic.

TABLE 4

SURVIVAL AFTER HOSPITAL DISCHARGE OF PREMATURES WITH BIRTH WEIGHTS OF 1,814 GRAMS OR LESS

Year Born	Infants Discharged from Hospital	Infants Known Dead before Age		% Known Deaths before 1 Year	Lost or Moved before 1 Year*
		6 months	1 Year		
1943	16	2	1	18.8†	2
1944	19	0	0		7
1945	30	1	1	6.7†	1
1946	49	0	0		5
1947	48	1	—	2.1	7
Total	162	4	2	3.7	22

* No record of deaths of these cases in city of Cincinnati.
† Two infants not included as deaths; one died at 13 and the other at 19 months of age.

POSTNATAL AND OTHER FACTORS

It has been stated that the factor (or factors) causing retrolental fibroplasia probably occurs in early postnatal life. The smaller premature infant is placed in an environment which, in spite of pediatric advances in handling, is far different from the optimum which would have been maintained in utero.

In general, the smaller prematures in this study received similar management. The policy in the premature nursery has been one of adapting treatment, such as the administration of intravenous and subcutaneous fluids, to the individual case rather than one of following a routine procedure. The environment, method of regulation of temperature and humidity, and administration

TABLE 5

MATERNAL FACTORS IN RELATIONSHIP TO RETROLENTAL FIBROPLASIA*

	Normal	Retrolental Fibroplasia	Significance
Bleeding:			
Present	16	1	None
Absent	65	5	$X^2=0.122$
Not stated	3	1	$P=>.70<.80$
Age of Mother at Delivery (years)			
20 or less	28	2	None
21–30	40	5	$X^2=1.708$
31 or over	12	0	For $n=2$, $P=>.30<.50$
Not stated	4	0	
Parity			
Primipara	31	0	None
Multipara	49	7	$X^2=2.682$
Not stated	4	0	$P=>.10<.20$
Virus Infection during Pregnancy			
Present	10	1	None
Absent	70	5	$X^2=0.856$
Not stated	4	1	$P=>.30<.50$
Chronic Illness			
Present	9	1	None
Absent	71	6	$X^2=0.748$
Not stated	4	0	$P>.30<.50$
Cause of Premature Onset of Labor			
Known	34	2	None
Idiopathic	47	5	$X^2=0.0832$
Not stated	3	0	$P=>.70<.80$
Race			
Colored	56	3	None
White	28	4	$X^2=0.734$
			$P=>.30<.50$

* Mothers of observed prematures with birth weights of 1,814 gm. or less, 1943 to 1948.

of oxygen and carbon dioxide have remained essentially the same during the 5-year period.

During the last three years, the diet has been changed from a usual one of breast milk or evaporated milk to a usual one of a formula relatively high in protein and low in fat according to the principles of Levine and Gordon.[9] The factor of type of diet showed no statistical significance between normal and retrolental fibroplasia cases.

During the last three years, increased amounts of vitamin K were administered following birth, but this also showed no statistically significant difference.

The infants routinely received a minimum of 2,000 units of oil-soluble vitamin D and a minimum of 100 mg. of ascorbic acid per day during their hospital stay. Neither specific medications nor the presence or absence of recognized illness during the hospital stay showed statistically significant differences. Three differences occurred, however, which warrant more detailed report.

VITAMIN A

Most of the infants received added vitamin A during their hospital stay. However, from May through December, 1947, water-soluble vitamin D without vitamin A was given. The only vitamin A these babies received was that included in the formula, approximately 590 units per day. This is less than the amount sometimes considered a minimum, although the actual requirements of vitamin A in the prematurely born infant are unknown.

Only the first month of life is considered in Table 6, since most of the babies had been discharged from the hospital before the age of two months. The choice of considering seven days as a dividing line was made because vitamin-A stores in the body are not so rapidly lost as are some other vitamins.[10]

TABLE 6

RELATIONSHIP OF VITAMIN A ADDED IN FIRST MONTH OF LIFE TO RETROLENTAL FIBROPLASIA IN PREMATURES WITH BIRTH WEIGHTS OF 1,814 GRAMS OR LESS, 1943 TO 1948

	Vitamin A Added 7 Days or More	Vitamin A Added 6 Days or Less*	Total
Normal	56	33	89
Retrolental Fibroplasia	1	6	7
Total	57	39	96

$X^2 = 4.4635$; $P = > 0.02 < 0.05$.

* In this group 30 prematures received no added vitamin A during the first month of life. The remaining three received oil soluble vitamin A as follows: (1) normal male, on the 30th postnatal day; (2) normal female, from the 26th to 30th postnatal day; and (3) male with retrolental fibroplasia, from the 27th to 30th postnatal day.

It could be expected that the administration of vitamin A for only seven days in the first part of the month would provide vitamin A for use in the body during the rest of the month. The difference in incidence between the groups given and not given additional vitamin A is not statistically significant.

All of the observed prematures in the series, who received vitamin A, were administered it in the oil-soluble form. Consequently, no comparison in results could be made between those receiving water-soluble and oil-soluble vitamin A.

SEX

In this survey, in the group of observed prematures with birth weights of 1,814 gm. or less 45.8 percent were males. Every case of observed retrolental fibroplasia was in a male. The predominance of the development

TABLE 7

RELATIONSHIP OF SEX TO OCCURRENCE OF RETROLENTAL FIBROPLASIA IN PREMATURES WITH BIRTH WEIGHTS OF 1,814 GRAMS OR LESS, 1943 TO 1948

	Boys	Girls	Total
Normal	37	52	89
Retrolental Fibroplasia	7	0	7
Total	44	52	96

$X^2 = 6.72$; $P = < 0.01$.

Identical twins were included in the table since affection of only one twin has been known. Only two pairs of identical twins, one boy and one girl, were observed in this weight group. If one member of each of these pairs is omitted, $X^2 = 6.77$; $P = < 0.01$.

of the disease in males in this study is statistically significant (table 7).

During the 5-year survey, only two prematures, both males, were administered testosterone (as an aid to bone maturation). Of these two, one developed retrolental fibroplasia and the other did not. Two cases are an insufficient foundation on which to draw any conclusions as to the role of testosterone. However, because of the fact that statistics indicate that retrolental fibroplasia is sex-linked, further investigation of the results of testosterone administration to prematures is indicated.

When the numbers of male prematures receiving additional vitamin A for seven days or more during the first month of life are compared with those who received less, the difference in incidence of retrolental fibroplasia is found to be not significant statistically (table 8).

TABLE 8

RELATIONSHIP OF ADDED VITAMIN A TO RETROLENTAL FIBROPLASIA IN MALE PREMATURES*

	Vitamin A Added 7 Days or More	Vitamin A Added 6 Days or Less	Total
Normal	20	17	37
Retrolental Fibroplasia	1	6	7
Total	21	23	44

$X^2 = 2.305$; $P = > 0.10 < 0.20$.

* Only first postnatal month and male prematures with birth weights of 1,814 gm. or less, 1943 to 1948, included.

TABLE 9

POSTNATAL DEVELOPMENT OF PREMATURE CHILDREN IN RELATIONSHIP TO RETROLENTAL FIBROPLASIA, 1943 TO 1948*

	Normal Development	Probably Normal	Retarded	Total
Normal Eyes	34	5	3	42
Retrolental Fibroplasia	4	0	1	5
Total	38	5	4	47

With probably normal considered as normal, $X^2 = 3.29$; $P = > 0.05 < 0.10$.

* All children included had birth weights of 1,814 gm. or less and were less than four years of age at time of examination.

ASSOCIATED INTRACRANIAL DISEASE

Dr. James Fisher[11] examined an unselected portion of the observed children with birth weights of 1,814 gm. or less and compared them with the Yale Developmental Scale. All were less than four years of age. His results are shown in Table 9. Only 1 of the 5 children with retrolental fibroplasia whom he examined was mentally retarded. The difference between the normal cases and the retrolental fibroplasia cases is not statistically significant in this group.

REPORT OF CASES

After a retrolental membrane has developed to its final extent and especially after the eye has become phthisical, it is difficult, clinically, to distinguish this condition from other pathologic conditions. Furthermore, at these stages the appearance may offer false or no clues as to the preceding course of events. Since routine ophthalmoscopic examinations from the time of birth to the development of retrolental fibroplasia have rarely been made, the descriptions of three cases are given in detail.

The normal eye in a newborn infant is very different in appearance from eyes of children who have reached the age of one year. Pigmentation, with the exception of most Negro babies, is usually clinically absent so that the irides are a grayish "infant" blue, and choroidal vessels are prominently seen in a reddish, hazy fundus. Discs have either no cupping, or slight elevation due to the presence of Bergmeister's papilla, and they are usually a dead white in color. Disc margins are frequently not distinct. The macula is not discernible until about the fourth postnatal month; this time is usually the same in prematurely born and full-term infants. Depending on the degree of prematurity, various degrees of persistence of the fetal vascular system are seen; in most cases these atrophy completely in the course of time.

CASE 1

History. This Negro boy, born on July 15, 1947, had a birth weight (at one-half hour of age) of 1,715 gm. (3 lbs. 12½ oz.). The mother, aged 25 years, had two full-term, normal living children, aged 6 and 7 years.

On June 4, 1947, she was admitted to a tuberculosis sanitarium with far-advanced tuberculosis of the lungs. During the pregnancy she had no history of uterine bleeding or virus infection. Kahn and Hinton serologic tests were negative, and she denied history of syphilis. She had no chronic illness other than tuberculosis. Delivery was precipitous and occurred after an estimated 7-month gestation. The mother died from tuberculosis 12 days after the birth of the baby.

The baby had no recognized illnesses during his hospital stay. Management was routine except that he received no vitamin A other than that contained in his formula for the first two months and evaporated milk for the remainder of his stay. He was discharged at the age of 3 months, 18 days, with a weight of 4,370 gm.

Eye Examinations. (Aged 10 days). External. O.U.: Lids, lacrimal apparatus, conjunctivas, corneas, pupillary size and reaction to light, and scleras were normal. Horizontal corneal diameters were 8.5 mm. Irides were pigmented.

Ophthalmoscopic. O.U.: Atrophic tags of the pupillary membranes were present. There was a good red reflex everywhere. The discs were pale, without cupping, and the margins were slightly blurred. No remnants of the hyaloid artery or tunica vasculosa lentis were seen. The retinas showed very early pigmentation. The retinal vessels were normal. There was no evidence of any retrolental fibrous tissue.

(Aged 3 weeks). External. O.U.: No change. Ophthalmoscopic. O.U.: The disc margins were now distinct, although there was no pigment outlining them. Otherwise no change.

(Aged 5 weeks). External. O.U.: No change. Ophthalmoscopic. O.U.: The color of the discs was now a normal pink and slight cupping could be seen. There was a slight increase in the retinal pigmentation. The retinal vessels, both arteries and veins, had become very tortuous; in the midperiphery many were corkscrew in shape. No aneurysmal dilatations were seen.

Since tortuosity of the retinal vessels is seen in

sickle cell anemia, a sickling preparation was made; there was no sickling.

(Aged 2 months, 11 days). External. O.U.: There was no change except for a possible faint whitish sheen in the pupillary area behind the lens.

Ophthalmoscopic. R.E.: With a plus 8D. lens, a pinkish gray tissue containing many fine blood vessels was seen in the lower half of the eye. This tissue had the clinical appearance of a detached retina. At the 6-o'clock position, projecting anteriorly from this tissue, there was a bright-red area of fresh hemorrhage. Above there was a gray area, which appeared to be thickened retina rather than detached retina; in this area no vessels could be seen, but the vitreous was hazy, obscuring it.

L.E.: Except for the area from the 10- to 12-o'clock positions a pinkish gray tissue could be seen behind the lens. From the 6- to 10-o'clock positions this was most clearly seen with a plus 10D. lens. The remainder was best seen with a plus 7D. lens. The tissue contained fine blood vessels and many fresh small hemorrhages. From the 10- to 12-o'clock positions, a gray tissue could be distinguished with a plano lens; the vitreous over this area was hazy. The interpretation was the same as that made for the right eye—thickened retina from the 10- to 12-o'clock positions and detached retina elsewhere.

In neither eye could the discs be seen. No elongated ciliary processes were seen. Tension to fingers was within normal limits. Tension was (local anesthesia, baby not crying): R.E., 25 mm. Hg with 5.5 gm. weight; L.E., 22 mm. with a 5.5 gm. weight (Schiøtz). The vessels of the retrolental membrane were not tortuous.

(Aged 3 months). External. O.U.: No change.

Ophthalmoscopic. R.E.: With a plus 10D. lens, there was a gray tissue containing fine blood vessels and old and fresh hemorrhages everywhere just behind the lens. L.E.: In the far periphery at the 8-o'clock position there was a little normal-appearing retina. Elsewhere there was a grayish vascularized tissue with fresh and old hemorrhages.

(Aged 3 months, 2 weeks). External. R.E.: For the first time a grayish white tissue with a few hemorrhages could be definitely distinguished filling the pupillary space behind the lens. L.E.: No change.

Ophthalmoscopic. R.E.: There had been no change except that the retrolental tissue had become whiter and appeared to be thicker. L.E.: The entire retina appeared to be detached, but the vitreous was so cloudy that details could not be seen.

(Aged 4½ months). External. R.E.: Tension to fingers was softer than normal. The pupil was bound down by a white secondary pupillary membrane with an irregular surface and could not be dilated with neosynephrin. L.E.: No change.

Ophthalmoscopic. R.E.: Examination could not be made. L.E.: Temporally, at the 9-o'clock position there was a grayish white tissue elevated 10 diopters. The vitreous was less hazy so that some retinal details could be distinguished. The retina was seen with a plus 1D. lens. The retinal vessels seen were not tortuous. On the surface of the retina extending forward into the vitreous, there were grayish white areas resembling either glial or connective-tissue proliferation. There were many fresh preretinal hemorrhages.

(Aged 5½ months). External. R.E.: In the center of the cornea there was a dense white opacity about 1.5 mm. in diameter. There was iris bombé. Tension to fingers was mushy soft, and the pupil would not dilate because of the secondary pupillary membrane. L.E.: No change.

Ophthalmoscopic. L.E.: The vitreous was so cloudy that undetached retina, if present, could not be visualized. There was an old hemorrhage behind the lens.

The foster mother reported that there were transient episodes of redness of the right eye during which time the baby would rub his eye and cry. At no time while the baby was under my observation was there either conjunctival injection or positive aqueous ray.

(Aged 7 months). External. Horizontal corneal diameters were 9.5 mm. A moderately rapid searching nystagmus had developed. R.E.: The corneal opacity had enlarged. Otherwise there was no change. L.E.: Tension to fingers was soft. Otherwise no change.

Ophthalmoscopic. L.E.: The vitreous was still very cloudy.

(Aged 8 months). External. R.E.: The corneal opacity was 5 mm. in diameter. In the upper temporal quadrant of the iris there was a brush of newly formed dilated vessels. L.E.: White tissue could be seen in the pupillary area. There was no pupillary reaction to light.

Ophthalmoscopic. L.E.: There was no red reflex. The dense white triangle of tissue at the 9-o'clock position was persisting. Elsewhere there was a grayish-white tissue elevated 10 diopters and containing fine blood vessels. A complete retinal detachment appeared to have occurred in this eye also.

The baby was sent to Boston to a home for preschool blind children. He was moderately retarded physically and mentally.

CASE 2

History. This white boy, born on November 3, 1947, had a birth weight of 1,715 gm. (3 lbs. 12½ oz.). The mother was aged 34 years at the time of delivery. She had one full-term, normal child, aged 5 years.

During this pregnancy she had had staining twice, once in the third month and once a week before the onset of labor. She had no history of virus infection, chronic disease, or syphilis. The Kahn test was negative. Labor occurred spontaneously at the end of an estimated 7½-month period of gestation, was of 3 hours' duration, and presentation was right occipito-anterior. There was no known cause for the premature onset of the labor.

At birth, the left side of the baby's face appeared bruised and blue. One week after birth the infant had a moderate jaundice. He had had a fracture of his 7th rib in delivery. On December 13, 1947, the baby developed gastro-enteritis due to an un-

known cause. The baby's management during his hospital stay was routine with the exception that he received no added vitamin A, other than that in his formula. He was discharged at the age of 2 months, 11 days, with a weight of 2,325 gm.

Eye Examinations. (Aged 3 days). External. O.U.: Lids, lacrimal apparatus, and conjunctivas were normal. Corneas were very slightly cloudy, probably due to silver nitrate administered at birth. Irides were an "infant" blue, or unpigmented. Pupillary size and reaction to light and scleras were normal.

Ophthalmoscopic. O.U.: Nonpatent pupillary membranes with three arcades were present. There was a good red reflex everywhere. Discs were the same shade as the retina which was clinically unpigmented. Disc margins were very indistinct, and there was no cupping. Retinal vessels were normal. No retinal hemorrhages, remnants of hyaloid artery or posterior tunica vasculosa lentis, or abnormal retrolental tissue were seen.

(Aged 2 weeks). External. O.U.: The corneas were quite clear. Otherwise no change.

Ophthalmoscopic. O.U.: The pupillary membranes had atrophied considerably so that only a portion of the first layer of arcades was well developed. Otherwise no change.

(Aged 1 month). External. O.U.: No change. Ophthalmoscopic. O.U.: Pigment was developing in the fundus so that the choroidal vessels were no longer plainly seen except in the periphery of the fundus. Otherwise no change.

(Aged 2 months). External and ophthalmoscopic. O.U.: No change.

(Aged 3½ months). External. O.U.: No change. Irides were blue but had lost their grayish, "infant" blue appearance. Tactile tension was normal. The anterior chambers were of normal depth, and no iris synechias were present.

Ophthalmoscopic. O.U.: The pupillary membranes had not changed in appearance. Nasally, in both eyes, there was a retrolental gray tissue containing small blood vessels and elevated 10 diopters. There were no hemorrhages on the surface of this membrane which extended over a fourth of the eye and appeared to be detached retina. Elsewhere there was a poor red reflex, but vitreous opacities inhibited a detailed examination of the retina. No elongated ciliary processes were seen.

(Aged 4 months). External. O.U.: No change. The corneas were 8.5 mm. in horizontal diameter. Ophthalmoscopic. O.U.: The nasal retrolental membranes had extended to the midline. Temporally, there was no membrane, but vitreous cloudiness obscured fundus details. Otherwise no change.

(Aged 4¾ months). External and ophthalmoscopic. O.U.: No change.

(Aged 5¾ months). External. O.U.: The retrolental membranes could be seen by focal illumination. Ophthalmoscopic. O.U. There had been a little clearing of the membrane in the far nasal periphery of the right eye. Otherwise no change.

(Aged 7 months). External and opthalmoscopic. O.U.: No change.

(Aged 7¾ months). External. O.U.: The baby usually held his eyes in a depressed position, but had no searching nystagmus. Both pupils reacted to light. Otherwise no change. Ophthalmoscopic. O.U.: No further change. Vitreous cloudiness persisted.

The baby's mental development was normal. He had still not attained the physical development of a full-term infant of comparable age.

CASE 3

History. This Negro boy, born November 23, 1947, had a birth weight (at one-half hour of age) of 1,086 gm. (2 lbs. 6 oz.).

The mother, aged 27 years, had had one abortion and had one full-term, normal daughter, aged 2 years. During this pregnancy she had no uterine bleeding or virus infections. In 1946, she was told that she had syphilis and she had 60 injections during that year. Her Kahn test was negative during this pregnancy, and there was no evidence of syphilitic infection of this baby. The mother had no other chronic diseases. Labor and delivery occurred spontaneously outside the hospital, after a 5½-month gestation, as estimated by the mother.

The baby was admitted to the hospital at the age of one-half hour. His development was poor; in the first month he gained only one ounce over his birth weight. On January 27, 1948, he developed atelectasis due to bronchial obstruction of right upper and lower lobes. Suction of the trachea and bronchi caused marked improvement.

His management was routine with two exceptions. From November 24th to December 20th he received testosterone (2.5 mgm., twice daily). He did not receive vitamin A, other than that contained in his formula until he was 49 days old. He was discharged at the age of 2¾ months with a weight of 2,380 gm.

Eye Examinations. (Aged 1 day). External. O.U.: Lids, lacrimal apparatus, conjunctivas, corneas, pupillary size and reaction to light, and scleras were normal. Irides were pigmented.

Ophthalmoscopic. O.U.: Completely formed pupillary membranes persisted. There was a good red reflex everywhere through dilated pupils. Detailed fundus examination was postponed, since the baby was in very poor condition.

(Aged 2 weeks). External. O.U.: No change. Ophthalmoscopic. O.U.: The pupillary membranes had atrophied slightly. A good red reflex was present everywhere. The discs were pale with no cupping but with distinct margins. The fundi were not clinically pigmented. Retinal vessels were normal. The maculas had not yet developed. No remnants of the hyaloid artery, or posterior tunica vasculosa lentis, or retrolental fibroplasia were seen.

(Aged 1 month). External and ophthalmoscopic. O.U.: No change except for further atrophy of the pupillary membranes.

(Aged 1 month, 2 weeks). External and ophthalmoscopic. O.U.: Fundi were pigmented. Otherwise no change.

(Aged 2 months). External and ophthalmoscopic. O.U.: Only slight tags of the pupillary membrane remained. Otherwise no change.

The patient's first appointment for examination after discharge and several others were broken because of his mother's illness; so he was not seen as frequently as desired.

(Aged 4½ months). A public health nurse had noticed an occasional nystagmoid movement the preceding week.

External. O.U.: No change. Horizontal corneal diameters were 10 mm. Pupillary size and reaction to light were normal. Anterior chambers and tactile tension were normal.

Ophthalmoscopic. R.E.: The pupillary membrane remnants had completely atrophied. There was a good red reflex everywhere. The disc had distinct margins, good color, and a little cupping. The macula had developed and was normal. Vessels and peripheral retina were normal. No retrolental membrane was present.

L.E.: Pupillary membrane remnants had atrophied. In the far temporal periphery behind the lens, there was a crescent-shaped hemorrhage bounded posteriorly by retinal detachment about one-half disc diameter in width and 10 diopters in elevation. Elsewhere, there was a good red reflex. The vitreous was clear; disc and retina were as in the right eye. The retinal vessels were not tortuous.

(Aged 6 months). The mother stated that she had been noticing a difference in the size of the two eyes.

External. O.U.: The horizontal corneal diameter of the right eye was 11 mm.; that of the left, 10 mm. Otherwise no change.

Ophthalmoscopic. R.E.: No change; normal. L.E.: In the far temporal periphery, hemorrhage was still present. The detached retina had become white in color, as if fibrosis and thickening had occurred, but the extent of the detachment had increased only slightly. The vitreous was still clear. Temporally, the retina contained many white streaks. The retinal vessels were not tortuous. The disc and nasal retina were normal.

(Aged 6½ months). External. O.U.: The left pupil did not react to light, but the right did.

Ophthalmoscopic. R.E.: No change. L.E.: The vitreous had become slightly cloudy. The temporal retinal detachment had increased. Temporally up to the disc, the retina was wrinkled and elevated 2 to 3 diopters. The nasal periphery was normal.

The mental and physical development of this infant was normal.

Discussion

The incidence of retrolental fibroplasia in this study appears lower than that found in Boston; however, since the percentage incidences of the Boston groups are the only figures cited, a statistical comparison cannot be made. A comparison with the incidence found by Dr. William and Dr. Ella Owens in Baltimore shows almost identical findings. In their observed cases with birth weights of 1,899 gm. or less, the incidence of retrolental fibroplasia was 6.0 percent. In this study, 7 cases of retrolental fibroplasia were found in 114 examined cases with birth weights of 1,899 gm. or less, an incidence of 6.1 percent (table 2. $X^2 = 0.00131$; $P = > 0.98 < 0.99$). In the observed groups in Baltimore and Cincinnati, the incidence of the disease is the same.

At the Cincinnati General Hospital there was a statistically significant increase in survival to hospital discharge of prematures with birth weights between 1,362 and 1,814 gm. from 1943 to 1948. However, this was not accompanied by a statistically significant increase in the number of cases of retrolental fibroplasia for the same period of time.

The incidence of retrolental fibroplasia in children given and not given additional vitamin A in the first month of life is not statistically significant in this study. However, since the probability of the observed difference being due to chance is less than 0.05, the figures indicate that a conclusive statement that vitamin A does not play a role in the development of retrolental fibroplasia in this study cannot be drawn.

The fact that in this survey more boys than girls developed retrolental fibroplasia is statistically significant. It evokes the question as to whether or not testosterone should be administered to prematurely born infants for therapeutic purposes.

Other possible etiologic factors were studied but were not found to have differences which were statistically significant. These factors were: uterine bleeding during pregnancy, age and parity of mother at delivery, occurrence during pregnancy of virus infections or chronic illness, cause of premature onset of labor, race, associated intracranial disease, vitamin A received by boys and early postnatal management and course other than those factors previously discussed.

The three cases of retrolental fibroplasia followed from birth have the following noteworthy findings:

1. External and ophthalmoscopic examinations of each baby during the first two weeks of life revealed no findings varying from those usually seen in the normal newborn premature infant. It is possible that at the very extreme periphery of the retina some pathologic process may have been present at birth. However, from the succeeding events this possibility would seem highly unlikely.

2. None of the three infants had a hyaloid artery or posterior tunica vasculosa lentis or their remnants present after the first two weeks of life. The cases observed from birth are too few to exclude the development of retrolental fibroplasia in eyes in which the hyaloid artery or posterior tunica vasculosa lentis or their remnants persist; however, it does exclude the suggested[2] role of these fetal vessels as structures producing the development of retrolental fibroplasia.

3. In one case, the first indication of any abnormality was the development of considerable tortuosity of the retinal vessels. The clinical appearance may resemble that seen in von Hippel's disease.

4. In each case, preceding the development of a retrolental membrane, elevation and detachment of the retina occurred. In two cases, hemorrhages were present on the surface of the detached retina, probably indicating some vascular disorder. After retinal detachment had persisted, the eye became white in appearance instead of gray, as if a little fibrous tissue had developed. The sequence of events in these cases seems to indicate that the retina is primarily involved in the formation of the membrane rather than secondarily involved, which was the earlier conception.[12, 13]

5. In all three cases varying degrees of vitreous cloudiness were observed. This may indicate the existence of an underlying retinitis or choroiditis.

6. In no cases, except after the development of shallow anterior chambers, synechias, and glaucoma, was there any injection of the conjunctival vessels or positive aqueous ray. This would seem, in the three followed cases, to indicate the absence of an anterior uveitis in the development of retrolental fibroplasia.

Elongated ciliary processes were seen only in the patients examined after the retrolental membrane was completely developed.

Summary

1. A survey for retrolental fibroplasia in 229 prematurely born children weighing less than 2,268 gm. at birth during a 5-year period was made in Cincinnati. No cases of retrolental fibroplasia were found in the group weighing above 1,814 gm. at birth. In 96 cases with birth weights of 1,814 gm. or less, 7 cases, or 7.3 percent, were found to have retrolental fibroplasia.

2. In this study the predominance of boys over girls among the infants affected with retrolental fibroplasia was statistically significant.

3. The following factors were considered in the etiology of retrolental fibroplasia: uterine bleeding during pregnancy, age and parity of the mother at delivery, occurrence of virus infections or chronic illness during pregnancy, cause of premature onset of labor, race, associated intracranial disease, administration of vitamin A in early postnatal life, and other factors in early postnatal management and course. The difference in the influence of these factors in affected and nonaffected prematures was not sufficient to be statistically significant and, therefore, the development of retrolental fibroplasia in this series cannot be attributed to them.

4. A description was given of three cases followed from birth until the development of retrolental fibroplasia.

2065 Adelbert Road (6).

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AMPUTATION NEUROMA IN THE ORBIT*

Frederick C. Blodi, M.D.
New York

Although true neuromas are rather uncommon, amputation or traumatic neuromas not infrequently develop at the severed end of a nerve trunk. These neuromas are really only a proliferation of the nerve fibers and of the connective tissue. They represent a clubbed overgrowth of coiled trunks at the extremity of the severed nerve. The local thickening may simulate a neoplasm.

When it was first proved that these amputation neuromas were not true neuromas, they were believed to be connective-tissue tumors, in which the nerve fibers play a minor and completely passive role. This theory (Goldmann,[1] Finotti[2]) assumed that only the connective-tissue fibers proliferate, while the nerve fibers, at the most, only elongate.

We know now that the connective tissue of the nerve is the first to proliferate. This connective tissue of the endo-, peri-, and epineurium is usually the main part of these neuromas. But into this mass of mesodermal tissue the nerve fibers push their way. The axis cylinders elongate, branch, and ramify into the connective tissue. As they are prevented by the surrounding tissue from growing straight forward, they form twisted bundles of medullated or nonmedullated fibers. The newly formed axis cylinders may be provided with myelin sheaths, because the nuclei of this sheath multiply also. The amputation neuroma, therefore, is essentially a regenerative overgrowth, similar to changes in simple regeneration of nerve trunks. Some authors nevertheless attribute to it a certain neoplastic character.

Amputation neuromas are always present after a certain time in an amputated limb. They are painful only when they are constantly pressed upon or pulled by the surrounding tissue. Not only the severed nerve of an extremity can give rise to such a neuroma, but also the cut nerves of the trunk, such as the intercostal nerves (Gouverneur[3]) and the sympathetic nerve

* From the Department of Pathology of the Institute of Ophthalmology, Presbyterian Hospital.

(Leriche and Fontaine[4]).

There are few reports of amputation neuromas in the orbit. This is surprising, since the nerves of the orbit are often cut in various operations. They should especially be expected to occur after enucleation of the bulb and after exenteration of the orbit. There is no reason why the ciliary nerves should be an exception in this regard. Apparently these neuromas are seldom the cause of neuralgias in the orbit, as they are not exposed to any mechanical trauma. Also histologic examinations of the orbit are rarely made after enucleation or exenteration. The histologic examinations which have been reported (Bock,[5] Sattler[6]) were performed too early after the operation to allow the development of an amputation neuroma.

Review of literature

The first case of an intraorbital traumatic neuroma was described by Bietti,[7] in 1900. He discussed the regeneration of the nerves after opticociliary neurectomies. In one of his histologically examined cases he found a retrobulbar neuroma in the scar tissue. It was composed of connective tissue and thick, mostly myelinated, intertwined nerve trunks, forming a nodule.

Ten years later Loehlein[8] examined histologically a series of patients after opticociliary resection performed because of pain or as a prophylaxis against sympathetic ophthalmia. In 2 of his 6 cases he found an amputation neuroma behind the globe.

Adamantiadis[9] found an amputation neuroma in the orbit five years after an operation for a hydatid cyst. This neuroma caused considerable pain and was the size of a big hazelnut. The nerve fibers of the neuroma were mostly myelinated.

Recently Babel and Valerio[10] reported the first case of an amputation neuroma after an enucleation. It caused neuralgia four years after the operation. The tumor was the size of a small nut and was connected with the optic-nerve stump. All the fibers were nonmyelinated.

Report of a case

A 3-year-old Negro boy, J. W., was admitted to the Institute of Ophthalmology on the service of Dr. A. B. Reese. For the previous 6 or 7 months, his grandmother had noted that the right eye looked peculiar. Except for the eye, examination showed the child to be healthy.

Ophthalmic findings. Right eye: The cornea was somewhat steamy. The anterior chamber was shallow and in its lower angle, in the region between the 5- and 6-o'clock positions, were some grayish white nodules. The iris was atrophic and several similar nodules were scattered over its anterior surface. Back of the lens was an orange reflex that occupied the whole pupillary area. The lens was clear, but pushed forward. The right globe was larger than the left one and showed a mild ciliary injection.

The left eye was normal; the fundus examined under general anesthesia showed no lesions.

X-ray films showed the typical mottled shadow of calcium density in the right orbit and none in the left. The optic canals were normal (Dr. R. L. Pfeiffer).

The diagnosis of retinoblastoma of the right eye was made and the globe with about 12 mm. of the optic nerve was enucleated. No implant was placed in the orbit.

The microscopic section revealed a retinoblastoma with total destruction of the retina. The choroid was invaded near the disc and near the ora serrata. Tumor cells were seen in the anterior chamber. The papilla of the optic nerve was densely infiltrated with tumor cells, which also grew through the lamina cribrosa. Additional islands of these cells were found farther behind the bulb in the optic nerve but the operative section was beyond the nerve invasion.

Postoperative course. Two months after the enucleation, there was noted in the apex of the right orbit a fullness which seemed to be somewhat more than could be accounted for by the muscle funnel. As the microscopic slides of the bulb did not show

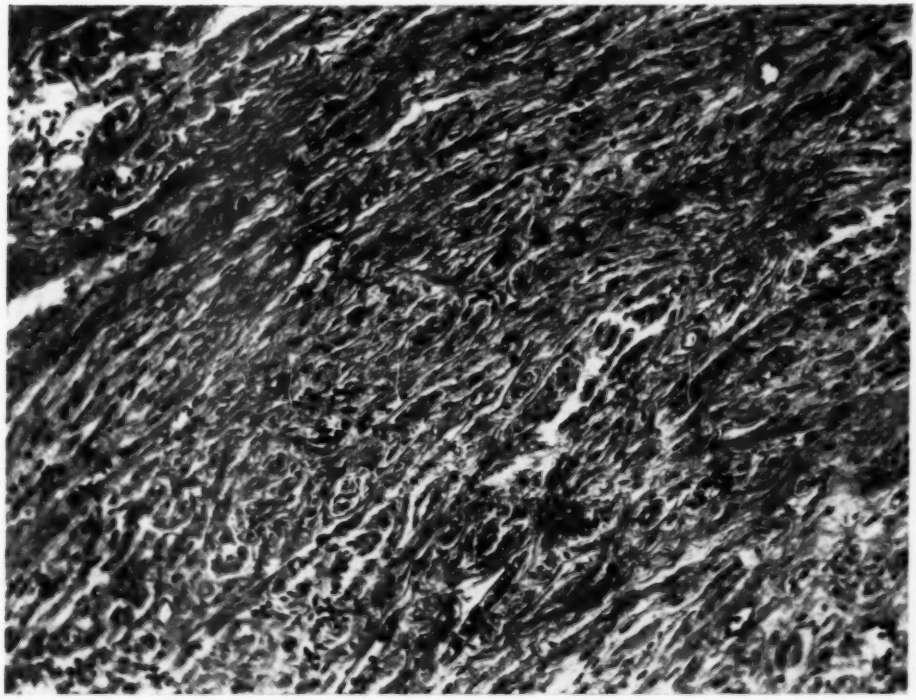

Fig. 1 (Blodi). Microphotograph of a section through the amputation neuroma.

any extraocular extensions and the operative section was beyond the tumor invasion of the optic nerve, a recurrence did not seem probable.

The boy was not back for observation at the appointed time and only returned again to the clinic four months later. At this time a large, firm, sharply demarcated, freely movable mass was palpable in the middle of the right orbit. This mass measured about 30 to 35 mm. in diameter and was not painful. It lay in the muscle funnel and was not attached to the bone or surrounding structures. No preauricular or cervical lymph nodes were palpable. A recurrence of the retinoblastoma was diagnosed and the boy was again admitted to the hospital.

An exenteration of the right orbit with skin graft was performed. The postoperative course was uneventful. A part of the specimen of the exenteration was prepared as a biopsy. It was the part of the tissue adjacent to the apex and it did not differ in consistency from the rest of the intraorbital contents.

The microscopic examination of the specimen revealed, to our great surprise, that this part was not a recurrence of the retinoblastoma, but an amputation neuroma (fig. 1). The nodule consisted of a mass of connective tissue and nerve fibers.

Centrally, the nerve fibers built up thinner trunks, which were densely interwoven with the connective-tissue fibers. These nerve trunks were twisted in all directions and crossed each other, so that on every slide they were cut transversely, longitudinally, and obliquely.

The connective tissue was dense and consisted of bundles of fibers, which were

similarly twisted. Toward the periphery of the nodule there was a preponderance of connective tissue which became denser and contained many capillaries and larger vessels. The nerve fibers were here arranged in larger trunks.

The nodule was poorly demarcated and had no capsule. It was surrounded by muscle tissue and by vessels. The muscle tissue was sharply demarcated from the nodule only in some places. In most parts the nerve fibers invaded the muscle tissue and spread among the muscle fibers, sometimes forming larger nerve trunks.

The major part of the intraorbital mass was prepared in the usual way. It showed a typical recurrence of a retinoblastoma. The tumor formed two nodules just beneath the conjunctiva. In the inferior periphery was a markedly thickened nerve running backward and becoming twisted. Apparently this was the place from which the biopsy was taken.

Discussion

The formation of an amputation neuroma in the orbit is certainly not as rare as is generally supposed. Such a neuroma might arise after any operation during which larger nerves are severed. In the orbit, it is presumably generally small and rarely causes neuralgic pain. The reason it is not more often found is that it does not cause symptoms and histologic examinations of the orbit after an operation are rarely performed. Amputation neuroma can easily be overlooked, especially in those cases in which the enucleation was not performed because of a malignant tumor and there is no reason to examine the orbit postoperatively. It is also probably difficult to palpate such a small tumor when there is an implant in the orbit.

In our case, the amputation neuroma reached a considerable size within seven months after the enucleation, which is rather early. Amputation neuromas have been recorded developing between 16 months and 54 years after an operation (Jedwabnick[11]). It may be that the youth of the patient and the keloid tendency of his race were related to the rapid growth of the regenerative process. The early detection of the tumor was due to the fact that the patient was watched for a recurrence after an enucleation for retinoblastoma.

Summary

The literature of amputation neuromas in the orbit is reviewed.

A case of amputation neuroma following an enucleation for retinoblastoma is reported.

635 West 165th Street (32).

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HERNIATION OF THE ANTERIOR HYALOID MEMBRANE FOLLOWING UNCOMPLICATED INTRACAPSULAR CATARACT EXTRACTION*

ALGERNON B. REESE, M.D.
New York

Following an uncomplicated intracapsular cataract extraction the anterior hyaloid membrane may herniate toward the anterior chamber. If the membrane rests against the cornea or pushes the iris forward, there may arise various sequelae which will be discussed in this paper. It should be emphasized that the observations given are based on intracapsular extractions with no complications at the time of the operation and with normal restoration of the anterior chamber.

At the time of the first dressing or later there may be a central, poorly demarcated, disc-shaped opacity of the cornea. The opacity is located around the posterior surface and is accompanied by wrinkling of Descemet's membrane and some edema of the stroma.

If the anterior chamber is extremely shallow or absent, the opacity may be more extensive and not confined to only the central portion of the cornea. An examination of these cases under magnification and preferably with a slitlamp reveals the hyaloid membrane to be in apposition to the cornea. The contact of the hyaloid membrane with the cornea is a cause of so-called "striate keratitis" following intracapsular cataract extraction.

In these cases, particularly if there has been a round-pupil extraction, a strong miotic such as D.F.P. (di-isopropyl-fluorophosphate) should be used. If the herination is large, the miotic may not accomplish a complete regression of the hyaloid membrane but may leave a collar-button-shaped knuckle extending through the pupillary area. In such a case the patient should be in bed, lying on the back, and the pupil should be dilated with neosynephrin (10 percent) and reconstricted with the miotic. These measures usually accomplish a complete regression of the membrane and a clearing of the corneal opacity, edema, and wrinkling of Descemet's membrane, as well as a prompt whitening of the eye.

If the case proves intractable, the patient should be put to bed lying on the back, the pupil should be dilated with neosynephrin, and air or Ringer's solution should be injected into the anterior chamber followed by the instillation of D.F.P. The latter is indicated in the presence of a round pupil in order to lock the hyaloid membrane out of the anterior chamber.

This type of "striate keratitis" does not occur in cases of round-pupil extraction so long as the pupil is well constricted. In these cases the condition appears after the first dressing when the pupil has been dilated with atropine or some other mydriatic. In order to overcome the atropine promptly, we have employed D.F.P.

A pupil which has been small may dilate when the herniation of the membrane occurs, and a pupil dilated by a herniated membrane may not easily constrict.

When the membrane touches the cornea, it usually causes some irritation of the eye, giving the clinical appearance of a very mild iridocyclitis. Besides the slight redness of the eye and the corneal changes, lacrimation and, to a lesser extent, photophobia are noted. This combination is often interpreted as a mild postoperative iridocyclitis and is treated by mydriatics, especially atropine. The use of miotics and other measures when necessary causes the condition to clear promptly.

The apposition of the hyaloid membrane to the corneal surface seems to disturb the endothelium sufficiently to permit aqueous

* From the Institute of Ophthalmology of the Presbyterian Hospital. Presented at the 84th annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, May, 1948.

to enter the cornea and thus produce edema of the stroma and overlying epithelium, particularly over the site where the hyaloid touches the cornea. Also, at the site of the apposition and in the region immediately adjacent to it, there is clouding and wrinkling of Descemet's membrane. If cornea guttata is present, so that the eye has the potentialities of an endothelial dystrophy, the apposition of the hyaloid causes more corneal changes than otherwise and may even lead to some permanent opacification.

Usually the herniation of the hyaloid membrane disappears spontaneously, and the membrane assumes a normal position in the pupillary area. When the membrane lies at the level of, or back of, the pupil, it usually shows a wrinkling. When it herniates into the anterior chamber, it usually presents a smooth, rather tense surface.

Kubik,[1] from an examination at the time of discharge of 526 cases of intracapsular extractions, found in 169 cases that the anterior hyaloid membrane projected flatly or in the form of a hemisphere. In the follow-up examinations he frequently could no longer see the protrusion of the hyaloid membrane. Vannas[2] could not give exact information regarding the frequency and rapidity of the recession of the herniation of the hyaloid membrane because he did not have a sufficient number of follow-up examinations.

In studying the anterior hyaloid membrane following intracapsular cataract extraction, there occurred an occasional instance in which a spontaneous rent in the membrane was noted months after the extraction. The membrane thinned out and, in the central portion where it was missing entirely, the vitreous protruded into the anterior chamber and over the surface of the iris.

Straight vitreous in the anterior chamber in these cases, or in those cases in which vitreous is lost at the time of operation, has not in my experience led to corneal changes.

From an examination of 32 eyes which had had intracapsular cataract extraction without loss of vitreous, Vannas found 10 cases with tears or holes in the anterior hyaloid membrane during a period of from 2 months to 4 years following the extraction. He felt that the hyaloid membrane more often showed a spontaneous rent in eyes in which the vitreous was diseased because a vitreous which became liquefied could manifest the same process by a weakening or even liquefaction of its hyaloid membrane. As most of the rents in the hyaloid membrane were located in the region of the pupillary margin, he speculated as to whether or not the action of the pupil could have had a slowly progressive traumatic effect.

Vannas found in 23 percent of the eyes with uncomplicated intracapsular cataract extraction that the anterior hyaloid membrane showed a concave surface and remained at the level of the pupil or even behind the pupillary border, and that in 61 percent of the eyes the height of the postoperative herniation of the anterior hyaloid membrane ranged from one fourth to one half the depth of the anterior chamber. In 9 percent of the eyes the anterior hyaloid membrane reached the posterior corneal surface. This author makes no mention, however, of corneal changes consequent to this herniation. His observations were made from the 7th to the 9th postoperative day.

Vannas also observed in his series of cases three instances in which during the period of observation with the slitlamp there was rapid alteration in the position of the anterior hyaloid membrane. For no apparent reason the membrane would alter its position from a state of herniation extending half the depth of the anterior chamber to a recession behind the pupillary border, forming a groove directed backward as deep as the hernia had been forward. He considered it possible that these fluctuations might be due to contraction and dilatation of the sphincter muscle of the iris, but that a more probable explanation was the state of contraction of the muscles composing the muscle funnel. He stated, however, that when these to-and-

fro movements of the vitreous are observed with the slitlamp, they seem to be entirely spontaneous.

The failure of the anterior chamber to restore, or the loss of the anterior chamber after it has been restored, following an intracapsular cataract extraction, may be due to advancement of the hyaloid membrane into the anterior chamber. Often a leaky wound is thought to be the cause of the absent chamber, but proof that this is not always the case is the fact that, if measures are taken to promote a retraction of the hyaloid membrane, the anterior chamber promptly restores, the corneal changes clear, and the symptoms subside.

An illustrative case is a round-pupil intracapsular extraction in which the anterior chamber was lost after it had been present for 12 postoperative days. Coincident with the loss of the chamber, there was increased redness of the eye, photophobia, lacrimation, and cloudiness of the cornea with wrinkling of Descemet's membrane. Examination with the slitlamp showed the hyaloid membrane against the posterior surface of the cornea. D.F.P. was instilled in the eye, the pupil contracted, the hyaloid membrane receded from the cornea, and, within five hours, the anterior chamber was restored, the cornea had cleared, and the symptoms had subsided.

After the cornea cleared, a choroidal detachment could be seen. The following day the pupil was dilated somewhat and the hyaloid membrane protruded but not so far as the cornea. D.F.P. was employed again and it has not been required since. The ability to influence a herniated hyaloid membrane by an intact iris is a distinct advantage of a round-pupil extraction. In cases of combined extraction the situation must be met by an injection of air into the anterior chamber.

There are rare instances in which recurrent herniations of the hyaloid membrane occur across the anterior chamber. These may take place any time, even up to years following the operation. When the membrane touches the posterior surface of the cornea, it causes a slight irritation of the eye with lacrimation, some photophobia, and even some discomfort or slight pain. The herniation may regress spontaneously, may recur, or may become permanent. These eyes usually show vitreous opacities and other indications of a very mild iridocyclitis which has apparently occurred as the result of the herniated membrane. Secondary glaucoma may ensue.

In one uncomplicated intracapsular cataract extraction there occurred even five years after the operation a slight congestion of the eye, vitreous opacities from a mild iridocyclitis, and reduction of the vision from 20/15 to 20/70. The only explanation I could find for this occurrence was that the anterior hyaloid membrane touched the cornea in the upper outer quadrant and produced corneal edema with epithelial stippling. The fact that there was some cornea guttata present may have aggravated matters. The hyaloid membrane became permanently adherent to the cornea over one area, and four years later the corneal edema and epithelial stippling had increased, some permanent scarring had ensued, and the intraocular pressure was elevated to 35 mm. of Hg (Schiøtz).

If the anterior hyaloid membrane does not retract spontaneously, or as the result of therapeutic measures, a permanent corneal opacity develops. This occurs because of the protracted period in which the membrane is in apposition to the cornea. Supposedly it may be facilitated by some trauma to the endothelium at the time of the operation. This trauma may be in the nature of corneal buckling when the lens is delivered, undue rubbing of the lens capsule across the posterior corneal surface at the time of the extraction or injuring of the endothelium with forceps or other instruments at the time of the operation. Thus, permanent scarring of the cornea ensues and the edema of the corneal stroma, as well as of the overlying epithelium, may persist indefinitely.

One case was observed in which the mem-

brane was in contact with the cornea for 3 months and caused corneal changes which cleared entirely when the membrane retracted. This eye was treated as a mild postoperative iridocyclitis and so atropine was employed. After the nature of the condition was appreciated, D.F.P. was used alternately with neosynephrin. The membrane retracted, the eye promptly whitened, and the cornea cleared.

When the membrane is permanently adherent to the corneal surface, the membrane may become thicker and denser adjacent to the site of the adhesion. This is thought to result from proliferation of the corneal endothelium over the surface of the adjacent hyaloid membrane.

If a permanent corneal opacity seems imminent in the pupillary area or adjacent to it, I think an attempt should be made to free the hyaloid membrane from the posterior surface of the cornea with a spatula. After this is done, air or Ringer's solution should be injected into the anterior chamber. If a round pupil is present, D.F.P. or some other strong miotic should be used with the patient in bed lying on the back. If the membrane cannot be freed from the cornea, I believe a deliberate rupture of the membrane would be beneficial.

Besides the usual type of iris prolapse* following cataract extraction, there is another type caused by the herniation of the anterior hyaloid membrane which pushes the iris ahead of it into the separated edges of the wound. The onset is insidious: it is not accompanied by pain and seldom by hyphemia. This may occur any time following the operation and tends not to be progressive. If operative measures are employed to correct it, an excision of the prolapsed iris is accompanied by little or no escape of aqueous and, behind the iris, can be seen the anterior hyaloid membrane herniating toward the wound. Unless care is taken, this membrane may be ruptured. There is some doubt in my mind as to whether or not this type of iris prolapse requires a surgical repair.

If the anterior hyaloid membrane herniates toward the operative wound over the site of an iris coloboma, there results merely some degree of separation of the wound lips. Frequently there is also some cloudiness of the cornea adjacent to the wound. Calhoun[3] believes this is sometimes confused with early epithelization of the anterior chamber. After a round-pupil, uncomplicated, intracapsular extraction the iris will sometimes develop a coloboma at one site some weeks to months following the extraction. In such cases there has been no iris prolapse but merely a gradual development of a coloboma. At least in some instances this is due to a herniation of the anterior hyaloid membrane which pushes the iris peripherally, causing some degree of inversion or recession.

Secondary glaucoma may ensue because the anterior hyaloid membrane herniates sufficiently at the periphery to advance the iris and block the angle, or because the membrane itself blocks the angle over the site of a coloboma of the iris. When the anterior chamber fails to reëstablish or is lost following the extraction due to the forward protrusion of the hyaloid membrane, glaucoma may occur because anterior synechias are produced.

In these cases the rational treatment seems to be the freeing of the membrane from the corneal surface with a spatula, a cyclodialysis operation with injection of air into the chamber, and the use of a strong miotic when a round pupil is present. If the membrane cannot be freed from the cornea by the above measures, I am not sure that it would not be wise deliberately to rupture the anterior hyaloid membrane.

* I refer to the type which occurs characteristically from the 4th to the 6th postoperative day and is caused by the fact that the wound cannot withstand the restored intraocular pressure and ruptures with escape of aqueous which pushes the iris into the wound. This is accompanied by sudden pain and usually by a hyphemia. If the prolapsed iris is not repaired, the prolapse becomes more extensive. An excision of the iris is followed by a gush of aqueous.

The glaucoma that may ensue following a round-pupil extraction may be due to the fact that the membrane protrudes in a pyramidal fashion through the pupillary area and thus causes the ball-and-valve effect described by Chandler.[4] The observation in one case supports this mechanism: in this case the hyaloid membrane herniated through the pupillary area but not so far as the posterior surface of the cornea. Glaucoma was present and there was an iris bombé so that, particularly in the periphery, the iris extended forward almost to the posterior surface of the cornea.

In such a case a dilatation of the pupil, followed if possible by a constriction of the pupil, may suffice to relieve the glaucoma provided it has not been of too long standing.

The incidence of glaucoma is said to be less following a simple intracapsular extraction than following a combined intracapsular extraction.[5] Following a combined extraction, the hyaloid membrane herniates more frequently and farther into the anterior chamber than following a simple extraction. This seems to be due to the fact that the intact iris offers support to the membrane. This, therefore, may be a factor in the lower incidence of glaucoma following simple extraction.

In order to establish the representative character of the cases from which these observations have been made, I have analyzed 100 consecutive cataract extractions performed by me just prior to writing this paper. This series includes, therefore, the complicated as well as the uncomplicated cases.

There were 10 glaucomatous eyes, 7 of which had had 1 or more filtering operations; 4 radiation cataracts; 2 eyes with old uveitis; 2 eyes with neurodermatogenous cataract; 1 cataract complicating retinitis pigmentosa; 1 cataract associated with detachment of the retina; and 1 cataract complicating malignant myopia. The complicated cataracts, therefore, were 21 percent. Ninety-two percent of the lenses were delivered in capsule. This includes 2 cases in which the capsule ruptured as the lens came through the wound. No capsule and no soft lens matter remained in the eye, and this was confirmed by the postoperative course. A third case in which the capsule ruptured as the lens came through the wound was not considered a delivery in capsule because some capsule remained in the eye and necessitated reëntering the anterior chamber to remove it.

Vitreous was lost at 4 operations, or 4 percent. Two of these were in the complicated group, one being a case of malignant myopia of over 20 diopters in which watery vitreous appeared at the time of the keratome section.

Discussion

The anterior hyaloid membrane normally extends from the base of the vitreous around the posterior chamber and makes contact with the lens at a site termed the "ligamentum hyaloidea capsularis." This membrane, which is much thicker than the posterior membrane, is a definite, rather dense, thick structure composed of a condensation of the vitreous elements. There is some histologic evidence to indicate that the membrane has a somewhat laminated structure. Outstanding characteristics of the membrane are its distensible and elastic properties.

At the time of a cataract extraction the membrane may bulge through the operative section. As the result of proper maneuvers, this herniation may retract and assume its normal or near-normal position. Following a contusion to the eye, the zonules may be ruptured and the anterior hyaloid membrane may herniate into the anterior chamber, the resultant appearance being one of a well-demarcated, globular mass which may recede or remain.

The anterior hyaloid membrane is normally in contact with the lens at the site of the ligamentum hyaloidea capsularis. It must be assumed that ordinarily this union is

merely one of apposition. Otherwise, if there were a firm ligamentous union, a rent would be torn in the anterior hyaloid membrane when an intracapsular extraction was done. There must, however, be variations in the degree of union between the membrane and the lens capsule. Even though in the majority of instances it may be merely one of apposition, there must be instances when an actual union of the two exists.

Thus, in these cases, when an intracapsular extraction is performed, the removal of the lens pulls away at least some of the lamellae of the anterior hyaloid membrane. If, then, this should occur, it may weaken the anterior hyaloid membrane and, therefore, predispose it to ectasia even under the normal vitreous pressure.

Likewise, there must be rarer instances in which a firm adhesion exists between the two. In these cases the removal of the lens causes an actual tear in the hyaloid, and this may account for the times when the delivery of the lens is followed by normal-appearing vitreous not under pressure.

For our discussion here, however, we are interested in the possibility that such a lens extraction may reduce the lamination of the hyaloid and thus weaken it. Clinically, evidence in support of this occurrence is the fact that under the slitlamp the thickness of the anterior hyaloid membrane following intracapsular extraction seems to vary considerably. In some instances it is even difficult to see. In others it is absent over an area where the vitreous is seen protruding into the anterior chamber through a rent or hole in the membrane.

Vannas found upon examination of 100 recent cases of intracapsular cataract extraction without loss of vitreous that there were 8 instances in which he could detect with the slitlamp a tear in the anterior hyaloid membrane.

It appears, therefore, that the anterior hyaloid membrane may be torn at the time of operation without loss of vitreous provided there is no vitreous pressure present. This tear or hole must be caused by an adhesion at the site of the ligamentum hyaloidea capsularis.

There are rare examples, particularly in complicated cataracts, perhaps as a result of inflammatory adhesion, in which there is a very firm adhesion between the posterior lens capsule and the anterior face of the hyaloid membrane. When such a union is firm, the surgeon appreciates it at the time of the intracapsular extraction because, as the lens comes forward, there is a feeling of an advancement of the entire vitreous body with it. In such an instance it may be necessary to peel or separate the hyaloid membrane from the lens capsule in order to remove the lens.

In the herniation of the anterior hyaloid membrane following an intracapsular cataract extraction, I believe an important factor is the change in the vitreous volume. It is necessary to assume such an occurrence to explain the transitory and even recurrent nature of the herniation. Lindner[6] claims that, following every cataract extraction as well as in all fistulizing eyes, there is a posterior detachment of the vitreous due to shrinkage.

The shrinkage of the vitreous may be due to aqueous replacement which in the aphakic eye is greater than in the phakic eye. Later, if the vitreous takes on fluid and regains its former volume, or even a portion of it, the anterior hyaloid membrane may protrude into the anterior chamber if the posterior vitreous detachment still persists.

It seems to me that a decrease in the lamination of the anterior hyaloid membrane (thus a thinning) as well as an increase in the vitreous volume must be assumed to explain the various manifestations of the herniation. The increase in the vitreous volume might explain the usual cases in which the hyaloid membrane protrudes straight through the pupillary area, but alone it would not explain the other cases in which the hyaloid membrane protrudes at a site away from the pupillary area. In these cases

it appears that a localized weakening of the membrane must also be assumed.

An instructive case was one in which the capsule broke after the lens had been dislocated over the lower one third. Postoperatively the patient developed a herniation of the anterior hyaloid membrane over the lower one third at the site where there was no remaining capsule. The membrane touched the cornea and produced a corneal opacity and edema at this site. The explanation must be that the anterior hyaloid membrane bulged at the site of least resistance when the vitreous volume was regained.

There are many who believe that, following a cataract extraction or a fistulizing operation, a choroidal detachment occurs secondary to a shrinking of the vitreous. If such is the case, the incidence of choroidal detachment should be less in extracapsular extraction than in intracapsular extraction. This is assumed because, when the vitreous regains its volume, the presence of the capsulozonular barrier prevents it from protruding into the anterior chamber. Therefore, it must expand posteriorly and decrease the posterior vitreous detachment, and thus the choroidal detachment. In other words, the anterior hyaloid membrane protrudes into the anterior chamber because this is the course of least resistance when the vitreous volume is replaced.

Von Sallmann[7] made some interesting observations on the behavior of the anterior hyaloid membrane following uncomplicated intracapsular cataract extraction. He noted that at the time when a detachment of the choroid is fully developed there is a definite herniation of the hyaloid membrane into the anterior chamber. When the choroidal detachment begins to subside, the bulging of the anterior hyaloid membrane recedes. This recession of the choroidal detachment and the recession of the herniated anterior hyaloid membrane go hand in hand until finally there is an actual depression or concavity of the hyaloid membrane behind the plane of the iris. This phenomenon was not noted in all cases of detachment of the choroid but only in those which regressed rapidly.

After the choroid becomes completely reattached, the recession of the anterior hyaloid membrane disappears, and the membrane assumes a normal position. The changes in the position of the anterior hyaloid membrane are coincident with those of the choroidal detachment and take place over a short period, the longest being several days.

Von Sallmann explains the recession of the previously protruding hyaloid membrane coincident with the reattachment of the choroid as follows: When the reattachment of an extensive detachment of the choroid proceeds rapidly, the regulation of the fluid absorption by the vitreous is not sufficiently rapid to prevent a temporary reduction in the vitreous volume. In this process the vitreous, which is adherent to the internal ocular structures, follows the receding choroidal surface, and this is manifested on the face of the vitreous by a retraction.

Lindner[6] integrates this phenomenon with his conception of a detachment of the choroid produced by a shrunken vitreous body in the following manner: The action of the vitreous on the choroid is generally horizontal, thus giving rise to the well-known manifestation of a choroidal detachment nasally and temporally. When the filtration of the operated eye, and therefore the abnormal production of aqueous, ceases, the pressure within the eye returns to normal. The vitreous thus tends to regain its normal volume. At this stage, however, any choroidal detachment present begins to recede, and the absorption of the subchoroidal transudate is apparently more rapid than the increase in vitreous volume, so that the anterior hyaloid membrane temporarily is retracted.

Further interesting observations were made by von Sallmann:[7] In a patient with secondary glaucoma following an uncomplicated intracapsular extraction, a cyclodialysis operation was performed and, at the time when the intraocular pressure was normalized as a result of the glaucoma op-

eration, he noted a herniation of the anterior hyaloid membrane into the anterior chamber.

Moderate pressure upon the eye through the upper lid caused the herniation to project farther forward, and fine wrinkles in the surface of the membrane disappeared. Following massage of the eye for one minute, the hyaloid herniation bulged farther and the membrane appeared more tense. Five minutes later the hyaloid membrane had receded behind the plane of the iris and showed wrinkling. Twenty-five minutes later the membrane had receded still farther behind the plane of the iris. One hour later the hyaloid membrane again herniated to the same degree to which it had before the massage was done.

The drainage of aqueous occasioned by the cyclodialysis operation ceased to function later, and the intraocular pressure rose to 50 mm. Hg (Schiøtz). The anterior hyaloid membrane lay behind the plane of the pupil. Pressure upon the globe through the upper lid caused the membrane to bulge markedly into the anterior chamber, but upon release of the pressure the hernia returned slowly to the level of the pupil. After massage of one minute the membrane still remained at the level of the pupil.

These observations indicated that, during the time in which the cyclodialysis operation was effective, the pressure upon the eye, as well as the massage, caused a definite increase in the extent of the already bulging hyaloid membrane. When the cyclodialysis operation became ineffective, the pressure and the massage on the eye produced no appreciable bulging of the membrane into the anterior chamber. Immediately following the massage, when the filtering operation was functioning, there was a rapid recession of the membrane. As a result of the communication between the anterior chamber and the suprachoroidal space, pressure and massage upon the eye forced the aqueous into the suprachoroidal space. As the aqueous from the anterior chamber decreased, the vitreous hernia could bulge farther into the anterior chamber. When the cyclodialysis was draining, the pressure and massage on the eye caused fluid to be forced out of the vitreous, and from 30 to 45 minutes was required for the vitreous volume to be replaced.

Summary

The contact of the anterior hyaloid membrane with the posterior corneal surface following uncomplicated intracapsular cataract extraction causes symptoms and signs which disappear if therapeutic measures are successful in causing the herniated membrane to recede from the corneal surface.

73 East 71st Street (21).

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Discussion

Dr. Edwin B. Dunphy (Boston): Dr. Reese's thorough presentation of this interesting subject has given us much food for thought. It emphasizes the harmful potentialities of the vitreous when this substance leaves its normal confines and spreads forward to new boundaries. Although many ophthalmologists are aware that vitreous in contact with the corneal endothelium for any length of time may give rise to edema of the

cornea, its occurrence in my experience has been extremely rare, considering the prevalence of vitreous herniation.

Dr. Reese apparently feels that this contact is the chief cause of the so-called striate keratitis seen postoperatively, although he mentions that it may be facilitated by damage to the corneal endothelium. It has always been my impression that damage to the endothelium at the time of operation is the *main* factor, since striate keratitis occurs just as frequently after extracapsular extraction where there is no possibility of vitreous touching the cornea.

It is my endeavor in intracapsular cataract surgery to deliver the lens by the Verhoeff sliding technique, the capsule being grasped at or near the upper equator with smooth forceps and pressure being made at the limbus below. A small iridectomy including the sphincter is performed before the delivery. The wound is closed by two Verhoeff corneoscleral tract sutures. By this method of extraction I feel that minimal damage to the cornea occurs.

The anterior chamber is reformed at the first dressing (24 hours). It is extremely rare to see any gross striate keratitis either then or subsequently, although slitlamp examination is not done until the 6th or 7th postoperative day, and it is possible, of course, that it might have been present earlier to a slight degree. At this examination vitreous is invariably found herniating through the pupillary space, sometimes practically in contact with the cornea, although not attached thereto. Wrinkling of Descemet's membrane is present in all cases, even when the hyaloid is nowhere near the cornea. In a few cases in which the reformation of the anterior chamber is delayed for a few days, I have not seen the saucer-shaped corneal opacity which Dr. Reese describes, although there is usually a lot of wrinkling of Descemet's membrane due to the hypotony.

I was interested in the quotation of Vannas's statistics to the effect that, in 23 percent of eyes with uncomplicated intracapsular extractions, the anterior hyaloid membrane showed a concave surface and remained at the level of the pupil, or even behind the pupillary border. In my uncomplicated cases I would estimate that practically 100 percent show some protrusion of the hyaloid in front of the pupillary border, in most instances extending halfway across the anterior chamber, and in some of them the vitreous is almost in contact with the cornea. Even when observed several years postoperatively this is true, although there tends to be a gradual retraction of the hyaloid. It is quite probable that, if I did round-pupil extractions, the incidence of extensive herniation would be less.

There is no doubt, of course, that corneal edema and opacification can be caused by adherence of the vitreous to the endothelium, particularly if the latter is damaged. Dr. Cogan of the Howe Laboratory, who has been making a special study of corneal edema, considers vitreous adherence to be an important contributory cause. Unlike Dr. Reese, he feels that the presence of the intact hyaloid is not essential; in other words, the edema may result from free vitreous in contact with the cornea following vitreous loss at operation. The condition of the endothelium is the determining factor. If it is in bad shape, either because of trauma at the time of operation or because of inherent degeneration, then the vitreous may stick to it with all the dire effects that Dr. Reese describes.

The injection of air to free the adhesion of vitreous to cornea has been attempted recently in one case at the Massachusetts Eye and Ear Infirmary. The patient, a man aged 68 years, had had intracapsular cataract extractions in each eye three years previously. Vitreous had been lost in one eye but not in the other. Both eyes showed vitreous herniating through the pupils and attached to the upper part of the endothelium at the line of incision. Edema of each cornea was present over the point of vitreous adherence. A shelving incision was made in the cornea

below just inside the limbus with a cataract knife. A good blast of air injected through this incision by a small needle and glass syringe succeeded in freeing the vitreous from the endothelium with subsequent clearing of the cornea to a large extent. The same process was successfully repeated in the other eye.

I am not familiar with the special type of iris prolapse caused by the vitreous pushing the iris into the edges of the wound as described by Dr. Reese, but see no reason why it should not occur, particularly in round-pupil extractions.

In similar fashion secondary glaucoma might be caused by vitreous pushing the iris root against the angle.

I have seen several cases in which the glaucoma was due to vitreous blocking the pupil according to the mechanism recently described by Chandler. In these cases there was a tendency to iris bombé, and a transfixion of the iris in the periphery relieved the situation by reëstablishing communication of aqueous between the posterior and anterior chambers.

The technique advocated by Dr. Reese of dilating the pupil, placing the patient on his back, and then constricting the pupil with a strong miotic might well be helpful in this type of case, particularly after round-pupil extractions.

The creation of a coloboma by the herniating vitreous pushing the iris peripherally has been observed by me on several occasions. A round pupil is not necessary for its production. Apparently in these cases the hyaloid ruptures, or thins out, not in the center of the pupillary zone, but to one side, so that the vitreous bulges through just behind the pupillary margin, shoving it toward the periphery. I have recently had such a case. During delivery of the lens some difficulty had been encountered in getting it to dislocate. Suddenly it gave way nasally, popping up into the wound. Extraction in capsule was completed without loss of vitreous. The next day the chamber was reformed and the operative result appeared to be perfect. On the third postoperative day I noticed a coloboma of the iris developing nasally. This increased slowly during the next three days. Slitlamp examination on the sixth postoperative day showed the picture just as Dr. Reese describes—the vitreous was bulging nasally, the adjacent pupillary border being displaced toward the periphery.

The author's supposition of a partial adhesion between the anterior hyaloid and the posterior surface of the lens to explain the weakening and bulging of the hyaloid following extraction is a reasonable one, and answers the question why this bulging sometimes takes place on one side and not in the center of the pupillary space.

The other factor he mentioned concerning the shrinkage of vitreous with choroidal detachment, and the correlation of changes in the position of the anterior hyaloid with the changes in vitreous volume, is an interesting one, and helps us understand the mechanism of vitreous herniation.

I have learned a lot from this paper, and am indebted to Dr. Reese for clearing up in my mind some things I never understood about herniation of the vitreous.

DR. F. H. VERHOEFF (Boston): I am much interested in this paper of Dr. Reese's and think it is a very important communication. I agree with most of the things he brought out, but it would take me about an hour to discuss all the aspects of the subject so I shall confine myself to just one, the permanent serious opacification of the cornea which results from adhesion of the vitreous to the back of the cornea. Some of us at the Massachusetts Eye and Ear Infirmary have known about this for at least 15 years. I would like to think I was the first one to get the idea, but my impression is that Dr. Gundersen is entitled to most of the credit.

For a good many years I had in mind a method of combating this opacification of the cornea but it was not until 1944 that I used

the method. I began to write up the case, but when I looked up the literature I could not find that anyone else knew anything about the condition, so it seemed foolish to report an operation for combating a danger that was almost unknown. I was, therefore, very much pleased to learn that Dr. Reese had established the fact that it was a serious condition with which I was dealing.

In March, 1944, I operated on the right eye of the patient, doing an intracapsular cataract extraction without loss of vitreous. She had 20/20 vision in this eye and wished me to operate upon the other eye, which I did three months later. She acted very well in the first operation, but in the second she was not a good patient, or at least that is to what I attribute the fact that I lost vitreous. The loss of vitreous was so slight that the pupil was not drawn up, but vitreous became adherent to the back of the cornea over a large area, about two thirds of the cornea. She began to get definite corneal opacification which, according to my experience, has been permanent and serious.

She was so well pleased with the right eye that she had the utmost confidence in me, very fortunately, otherwise I could not have done what I did for the left eye. She had edema of the cornea and opacification, so I decided that eye would be ruined if I did not do something. I made a keratome incision, let out all the vitreous that would come, and then I even put an irrigating tip in the anterior chamber, but irrigation did not bring out much more vitreous. I used only one suture this time. The wound healed very well and the anterior chamber was free of vitreous. The cornea cleared up except for a slight opacity high up, which is there today. But, as usually happens after a primary large loss of vitreous, the pupil became drawn up so high that one could hardly see it, and the eye remained irritable for months. The eye therefore had very little vision at that time. The patient stuck by me all this time in spite of photophobia and discomfort. At the end of nine months, when the eye had become white and quiet, I did a small iridotomy with a Ziegler knife and obtained 20/30 vision, which she still has.

I suspect that a slight loss of vitreous is more likely to produce the condition than a large loss. After a large loss I think most often the vitreous goes back to the level of the pupil. I do not think it is necessary for the hyaloid to be in front of the vitreous. It is the contact of the vitreous with the cornea that produces the opacification. Dr. Cogan thought possibly opacification would be more likely to occur if there was cornea guttata. Cornea guttata was present in both eyes of my patient. I have not had many of these cases myself, but I have seen the condition a number of times in eyes operated upon by other surgeons.

I recall one case especially in which I wish I had done this operation of vitreous removal. The patient had been operated upon in Hartford by one of the members of this society and had opacification of the cornea. I thought the operation must have been badly done, and I would do a better one, so I operated on the other eye and had exactly the same thing happen. The man was a very "bad actor"; I lost vitreous and I imagine my colleague in Hartford did also. Both eyes had such opacification of the corneas that the patient could only count fingers. I feel sure that if I had removed the vitreous from the anterior chamber before serious opacification of the cornea had occurred, this patient would have obtained good vision.

Dr. William P. Beetham (Boston): I would like to say a few words in behalf of the injection of air into the anterior chamber at the time of cataract extraction. Some of us in Boston have been somewhat disappointed with the injection of air into the anterior chamber in some of the late cases of this complication which we are talking about. By late cases I mean those with serious corneal damage in which adherence of the vitreous to the posterior surface of the

cornea has occurred at a rather late date following cataract extraction. The vitreous, either free or with the hyaloid intact, can be pushed back from the posterior surface of the cornea, but usually by the time this is done the corneal changes are severe and irreversible, and the situation proceeds, namely, edema, bullous keratitis, and corneal opacities.

Since vitreous prolapse can occur postoperatively from hours to days following operation, it does not seem reasonable that anyone can predict which eyes will be affected, and when. The chances are that, when these people appear for treatment, they will have seriously damaged corneas or irreversible changes. In contradistinction to that group for which little can be done are the early cases, for which I think considerable can be done, namely, prevention, and by prevention I mean preventing the occurrence of the adherence of the vitreous to the posterior surface of the cornea by doing something at the time of operation.

I think at this point we ought to thank Dr. Reese for admirably describing this condition and bringing it to our attention. For 6 or 7 years I have made it my routine procedure to inject not a bubble of air but if possible to fill the anterior chamber with air at the time the cataract is removed through my iridodialysis opening after two Verhoeff stitches have been tied.

Last week I inspected the records of 300 operations done during the last three years, and I found one case which fufilled Dr. Reese's description. That patient was one in whom striate opacity was noted at the first postoperative dressing. It has persisted for two years with a maintained acuity of 6/12; vitreous is still adherent to the posterior surface of the cornea. In my description of the record of that operation, believe it or not, I make the statement "no air was injected into the anterior chamber." Why, I do not know, but I operated on the other eye of that patient two years before that operation and air was injected into that anterior chamber, and she had a good result, so it is interesting to me that the eye in which air was presumably injected did not develop this complication.

My point is merely this, that for some time I have believed that the injection of air into the anterior chamber immediately following cataract extraction is an important addition to the modern cataract operation, and I think Dr. Reese's paper has made me feel more decisive about it.

Dr. Charles A. Perera (New York): I wish to agree with Dr. Dunphy in his remarks in regard to the cause of striate opacification. I think Dr. Reese's thesis on herniation of the anterior hyaloid membrane is only one of the causes, and perhaps not the main cause in most cases. I wish to report an example of this condition following a cataract operation with round pupil and a peripheral iridectomy. This was done on an 81-year-old man; the postoperative course was uneventful, and air was injected into the anterior chamber at the close of the operation as Dr. Beetham has done for years. The night after the operation the patient got out of bed and wandered about until he was finally put back to bed.

At the first postoperative dressing the anterior chamber was filled with blood which extended under the conjunctival flap. When the blood was sufficiently absorbed a small iris prolapse was seen temporally; the cornea in this quadrant showed striate opacification and the blood-filled vitreous pressed against the cornea in this quadrant. After consultation with Dr. Reese, and in accordance with his suggestion that air injection might be worth while, I performed this procedure as Dr. Dunphy described it. The vitreous was pushed back from the cornea, the corneal opacification rapidly disappeared, and the vitreous is now clearing.

Dr. Ralph O. Rychener (Memphis): I enjoyed Dr. Reese's paper and its very timely explanation of a number of things which I have been puzzled about, and I want to add a little confusion to it. With regard

to air injection immediately following intracapsular cataract extraction for uncomplicated cataract, in the past two years I did that routinely, employing the method suggested by Dr. MacMillan in which I put in a lot of air, enough to depress the iris, and it was during that period I ran into all the complications of herniation of the vitreous against the endothelium which I have encountered in my limited practice. So after reviewing my operations I decided perhaps the air was responsible for the opacification of the cornea and have now stopped air injection. I would like to ask Dr. Reese, when he dilates with neosynephrin and employs strong miotics, how long does he employ the miotic, and what happens after he stops it?

Dr. Alfred Cowan (Philadelphia): I have been interested in this subject for many years, and particularly in the acceptance of the term "anterior hyaloid membrane." In 1931, I presented a paper before this society describing the anterior hyaloid membrane; in 1932, Dr. Fry and I gave what we thought was conclusive evidence that there is such a thing as an anterior hyaloid membrane and, in 1939, before this society again, Robb McDonald and I presented a report of a series of cases of postoperative aphakics.

In this series was a group in which the lens and its capsule were supposed to have been extracted—intracapsular extractions. In nearly every one of these cases, immediately after the operation, the anterior hyaloid protruded far into the anterior chamber, and in many instances it was in contact with the posterior surface of the cornea over quite a large area. I should say in most of the cases we examined protrusion of the hyaloid into the anterior chamber persisted until the 5th or 6th day.

After that—now I am describing those in which the operation was successful, in that the hyaloid membrane was intact—after that, the hyaloid retracted gradually and, in the majority of instances, later on it would lie in the plane behind the iris without anything having been done for it. It seems to me after all this time (and I have studied a great many of these conditions) that it is not a matter of pressure from behind that causes the hyaloid to protrude but rather it is due to a lack of pressure or support in front.

As far as striate keratitis is concerned, I think it is possible that herniation of the hyaloid is a factor; but I do not think it is an important factor, because in almost every case after you open the globe you will find some indication of folds in Descemet's membrane or in the posterior portion of the cornea. I should like to show a slide which contains illustrations from an article by me and of one by McDonald and me. The first drawing illustrates a case that can be considered a narrow slitlamp appearance of the hyaloid membrane which is concave from before backward and lies behind the plane of the iris. This is what is considered a successful extraction of a lens with its capsule after 6 or 7 days. There is a drawing of a case in which a permanent herniation resulted; it is hemispheric. This is not a rare result. Another represents an instance in which the hyaloid lies in horizontal folds. One drawing illustrates a case in which blood in the vitreous is contained in the hyaloid which protrudes into the anterior chamber over the lower border of the pupil. After the blood became absorbed, the hyaloid membrane contracted behind the plane of the iris. Another is an illustration of an instance in which the hyaloid became incarcerated in the wound. It seems to me that such cases are not due to pressure from the force behind but that the hyaloid is drawn into the wound as the knife is withdrawn.

Dr. Arthur J. Bedell (Albany, New York): I wish to show one slide which has some historical interest because it was exhibited almost a quarter of a century ago. You can see the vitreous prolapse in each drawing. Dr. Reese told me he had not seen the article which was presented before the English-speaking Congress in 1925. It is for that reason I present it to those who may be unaware of this early work.

DR. ALGERNON B. REESE (closing): Dr. Dunphy mentioned the fact that he and also Dr. Cogan feel that the condition of the endothelium is an important factor in the corneal changes. I agree with them. I mention in the paper that if cornea guttata is present the corneal changes are more severe.

Dr. Dunphy also mentions the fact that herniation of the hyaloid may produce a coloboma of the iris. This point is also discussed in the paper.

A feature of Dr. Dunphy's and Dr. Verhoeff's discussion with which I am not entirely in accord is that dealing with the ill effects of straight vitreous on the cornea. I am not sure that vitreous alone produces corneal changes. I think the changes I have observed have occurred as the result of the hyaloid membrane. This membrane has a definite structure and it is the effect of this against the cornea which promotes symptoms and signs.

I was glad to hear Dr. Verhoeff state that he felt a deliberate rupture of the hyaloid membrane is indicated sometimes. I have thought that this might be beneficial sometimes in cases with permanent adhesion of the membrane to the cornea, and in cases with secondary glaucoma.

Dr. Beetham mentions the value of air injections. I think they have value not only in pushing the iris back but in pushing the hyaloid back. I recall the case which Dr. Perera quoted, and I feel, as he did, that the injection of air was beneficial.

Dr. Rychener asks how long miotics are used. I think a period of about 2 to 3 days is adequate.

I know of Dr. Cowan's work. I referred to the hyaloid as a membrane which is in agreement with his ideas. I thank Dr. Bedell for calling my attention to the article which he wrote in 1925 pertinent to this subject.

OPHTHALMIC MINIATURE

Blumen

(Dem Augenarzt von seinen Kranken.)

Sie kommen aus dem Schoss der Nacht:
Doch waren unten sie geblieben,
Wenn nicht das Licht mit seiner Macht
Hinauf ins Leben sie getrieben.

Holdselig aus der Erde bricht's,
Und blüht nun über allen Schranken;
Du bist der Freund des holden Lichtes:
Lass dir des Lichtes Kinder danken!

Theodor Storm (1817-1888).

THE ROLE OF CILIARY AND SUPERIOR CERVICAL GANGLIA IN OCULAR TENSION*

ERNST SCHMERL,† M.D., AND BERNHARD STEINBERG, M.D.
Toledo, Ohio

This study is a part of a general investigation of the factors which affect intraocular pressure. In a previous report it was indicated that the diencephalon contained a center or centers which exercised a regulatory control of intraocular pressure.[1] In a subsequent paper[2] it was shown that the central control of tension involved the pituitary body. The presence of two active principles, probably hormonal in nature, was found in the region of the posterior lobe of the pituitary. One of these principles increases and the other decreases the tension. In a normal state they are in an equilibrium. Apparently, these active principles are secreted into the spinal fluid and they act upon the diencephalic center or centers.

It was postulated that stimuli which are induced in the diencephalon are transmitted through the medium of the autonomic nerve fibers to the eyeball. These stimuli dilate or constrict capillaries depending upon the predominance of either of the two principles. There is ample clinical and experimental proof that capillary action represents one mechanism which determines ocular tension.[3]

The present study concerns itself with the role of the ciliary and superior cervical ganglia in ocular tension and the postulated mechanism which is outlined in this paper.

Saitoh[4] extirpated the ciliary ganglion and found an immediate decrease of intraocular pressure which returned to normal in an hour. The tension was low again the second day and returned to normal within two weeks. Peschel[5] described a procedure for removal of the ciliary ganglion and noted a decrease in tension. Givner,[6] among others, pointed out the occurrence of accessory ciliary ganglia. Linksz[7] observed the effect of the sympathetic pathways on ocular tension.

EXPERIMENTAL PROCEDURES

Rabbits were used as experimental animals. The ciliary ganglion was reached by a bipolar electrode which was insulated except for the tip. The electrode could either stimulate or cauterize. The tip of the electrode was introduced through an incision in the anterior lower part of the eyelid after removal of the tear and Harder glands. The electrode was guided by the trigeminal nerve to the region of the ciliary ganglion. Contact with the ganglion was recognized by contraction of the pupil and a rise in the ocular tension after stimulation. Stimulation was followed by cauterization. The method was not universally successful. Only those animals were used in the experiments in which the pupils were dilated, the pupillary reflex was absent, the wink reflex was retained, the corneal sensation remained intact, and the fundi were normal.

The left ciliary and the superior cervical sympathetic ganglia were stimulated and cauterized in 10 rabbits. The ocular tension was determined prior to the action upon the ganglia and immediately after stimulation and up to 21 days following cauterization. In 4 animals with cauterized ciliary ganglion, ocular tension was determined after administration of distilled water for 60 to 90 minutes.

In 7 rabbits, following either cauterization of the ciliary ganglion or excision of the superior cervical, spinal fluid, in volumes of 0.5 to 0.8 cc., of animals exposed to dark or to light was injected. The tension was determined for 60 to 90 minutes following the injection. Normal animals were injected with identical spinal fluid and were used as controls. As in our former studies, changes

* From Toledo Hospital Institute of Medical Research. This work was supported by a grant from the Snyder Ophthalmic Foundation.

† Research ophthalmologist of the Snyder Ophthalmic Foundation.

in tension of 3 or more mm. Hg (Schiøtz) were considered significant.

Results

Stimulation of the ciliary ganglion in 5 animals resulted in an immediate increase of intraocular pressure of from 8 to 17 mm. Hg (table 1). Other changes consisted in pupillary contraction, marked hyperemia of the iris, flattening of the anterior chamber, and haziness of the iris. These changes are indicative of stimulation of the parasympathetic fibers of the oculomotor nerve with a resultant capillary dilation and increased permeability.

TABLE 1

Effect of faradic stimulation of the ciliary or superior cervical ganglion upon intraocular pressure

No. of Rabbit	Area Stimulated	Ocular Tension in mm. of Hg in Relation to Stimulation	
		Before	After
1	Ciliary Ganglion	16	33
2		20	31
3		21	31
4		17	33
5		28	36
6	Superior Cervical Ganglion	25	14
7		21	14
8		23	16

Cauterization of the ciliary ganglion in 6 animals resulted in a decrease of intraocular pressure of from 8 to 13 mm. Hg (table 2). The readings were taken at intervals of from 1 to 28 days. Generally, the ocular tension showed a tendency to slight recovery after several days. Other changes which were observed consisted of pupillary dilatation and loss of light reflex.

Cauterization of the ciliary ganglion followed a month later by ingestion of 40 cc. of distilled water per kilo body weight resulted in little or no change. Under normal conditions, with an intact ciliary ganglion, the tension rises (table 2).

Cauterization of the ciliary ganglion followed 5 to 7 months later by intravenous injection of spinal fluid from animals kept in the dark showed a reduction of ocular tension equal to that found in normal animals (table 3). The reduction occurred within 15 to 20 minutes and persisted for the subsequent 30 to 60 minutes. Injection of spinal fluid from animals exposed to light showed no change. On the other hand, the intraocular pressure was elevated in the control animals (table 3).

These changes indicate that removal of the parasympathetic pathway results in a failure to transmit those stimuli which cause an increase in tension. At the same time, there is indication that there is no interference with the active principle which causes a decrease in tension.

Stimulation of the superior cervical ganglion resulted in a lowering of the intraocular

TABLE 2

Effect of cauterization of ciliary ganglion upon intraocular pressure

No. of Rabbit	Ocular Tension in Relation to Cauterization			Ocular Tension in Relation to Cauterization after Administration of Distilled Water		Ocular Tension of the Eye with Intact Ciliary Ganglion in Relation to Water Intake	
	Before	After		Before Water	After Water	Before	After
		1 Day	14–28 Days				
1	16	8	15	14	14	18	22
1	—	—	—	15	10	17	18
2	21	12	18	18	19	18	27
3	17	4	17	17	21	19	27
4	21	9	12	12	14	17	22
5	20	9	10	—	—	—	—
6	28	16	—	—	—	—	—

TABLE 3

EFFECT UPON INTRAOCULAR PRESSURE AFTER CAUTERIZATION OF LEFT CILIARY GANGLION FOLLOWED BY INJECTION OF SPINAL FLUID FROM RABBITS EXPOSED EITHER TO LIGHT OR DARKNESS

No. of Animal	Left or Right Eye	Type of Spinal Fluid	Ocular Tension in Relation to Injection of Spinal Fluid		Control Animals with Intact Ganglion and Injected with Spinal Fluid	
			Before	After	Before	After
1	R	From **Light**-Exposed	18	18	21	25
	L	Animals	15	15	21	24
2	R	"	18	18	25	28
	L		18	18.5	25	28
3	R	"	18	18	26	30
	L		14	15	26	28
4	R	From **Dark**-Exposed	16.5	14	19	15
	L	Animals	16	12	19	17
5	R	"	19.5	16	21	17
	L		19.5	16.5	21	18
6	R	"	21	18	—	—
	L		19	16	—	—

pressure of from 7 to 11 mm. Hg (Schiøtz).

Removal of the superior cervical ganglion followed 5 to 6 months later by injections of spinal fluid from animals exposed to light showed an increase in tension of from 3 to 9 mm. Hg. Injection of spinal fluid from

TABLE 4

EFFECT UPON INTRAOCULAR PRESSURE AFTER REMOVAL OF LEFT SUPERIOR CERVICAL GANGLION FOLLOWED BY INJECTION OF SPINAL FLUID FROM RABBITS EXPOSED EITHER TO LIGHT OR TO DARKNESS

No. of Rabbit	Left or Right Eye	Type of Spinal Fluid	Ocular Tension in Relation to Injected Spinal Fluid		Ocular Tension in Control Animal with Intact Ganglion in relation to Injected Spinal Fluid	
			Before	After	Before	After
1	R	From **Light**-Exposed	25	28	—	—
	L	Animals	25	28	—	—
2	R	"	26	29	—	—
	L		26	30	—	—
3	R	"	20	27	21	25
	L		20	29	21	23
4	R	From **Dark**-Exposed	24	25	19	16
	L	Animals	25	25	20	17
5	R	"	25	24	19	15
	L		25	26	19	17
6	R	"	20	21	21	17
	L		22	23	21	18
7	R	"	22	22	25	20
	L		21	23	25	21
8	R	"	24	24	24	20
	L		24	27	23	19

rabbits exposed to darkness showed no significant change in tension (table 4). These changes indicate that removal of the sympathetic pathway results in a failure to transmit those stimuli which cause a decrease in tension. At the same time there is indication that there is no interference with the active principle which causes an increase in tension.

Summary

Under the conditions and on the basis of these experiments, it may be postulated that stimuli travel from the diencephalon by way of the ciliary ganglion, which is essentially of parasympathetic nature, and produce vascular dilatation with a consequent increase in ocular tension.

On the other hand, those stimuli from the diencephalon which travel by way of the superior cervical ganglion, a sympathetic conveyer, contract capillaries and produce a lowering of intraocular pressure. Light and darkness incite the production of active principles in the pituitary. These principles act upon the diencephalon and initiate nervous stimuli which are transmitted to the periphery along the autonomic pathways.

2805 Oatis Avenue (6).

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Ophthalmic miniature

Celsus contains a summary of all that was known in his time. Although he was ignorant of the seat of cataract, he has described the operation of couching excellently and concisely, not omitting the important subjects of previous preparation and after treatment, for which his directions are judicious.

Sir William Lawrence, *A Treatise on the Diseases of the Eye*, 1833.

THE EPIDEMIOLOGY OF EPIDEMIC KERATOCONJUNCTIVITIS*

PHILLIPS THYGESON, M.D.
San Jose, California

The recurrence of epidemic keratoconjunctivitis in California in epidemic form in 1947 and 1948 indicates that this disease, which first became widespread in the United States in 1941, is probably to be a permanent problem. Although it failed to have the expected military significance during the war years, it did become an industrial problem and would seem to be potentially capable of causing major disturbances in the future.

In view of the office transmissions which occurred in both the 1941-1942 and the 1947-1948 epidemics, and in the course of which many ophthalmologists became infected, it would seem pertinent at this time to reëxamine the epidemiology of this important disease. The present clinical study is based on my personal observations, on the observations of a number of colleagues, and on epidemiologic studies previously recorded in the literature.

CLINICAL CHARACTERISTICS

The clinical characteristics of the disease as it appeared in the 1941-1942 epidemic have been well described by Hogan and Crawford,[1] Braley,[2] and Holmes.[3] My own experience has been limited to approximately 90 cases about equally divided between the two epidemics. In these cases the disease was characterized by an acute onset with preauricular adenopathy, by the nonpurulent character of the exudate, and by the development in most cases of small, round, subepithelial corneal opacities after an interval of from 7 to 10 days.

The conjunctival involvement varied from a follicular hypertrophy in mild cases to a severe pseudomembranous, or occasionally membranous, conjunctivitis in severe cases. The corneal complications varied similarly from mild involvement without visual impairment to severe involvement with reduction of vision to 20/200 or even less. The severity of the corneal involvement did not always parallel the severity of the conjunctival involvement.

In most cases the vision eventually returned to normal but occasionally the lowered acuity persisted. This was apparently related to the diffuseness of the infiltration since persistent round infiltrates were often associated with normal vision.

There were a number of severe cases in which multiple petechial hemorrhages in the skin occurred. Coincidental respiratory symptoms were observed in a small percentage of cases and the complaint of headache at onset was common enough to suggest the possibility of meningeal irritation. In the 1947-1948 epidemic in California there was a noteworthy degree of mental depression among the patients during the first few weeks of illness.

The clinical diagnosis of epidemic keratoconjunctivitis can be made with certainty only after the development of the characteristic round, subepithelial infiltrates, but a presumptive diagnosis can be made on the basis of the clinical picture of an acute follicular conjunctivitis with grossly visible preauricular adenopathy, combined with the laboratory finding of a mononuclear cell exudate without significant bacteria.

Differential diagnosis must be made from the following disease entities: (1) Acute herpetic keratoconjunctivitis, (2) acute follicular conjunctivitis (Beal), (3) nummular keratitis (Dimmer), (4) inclusion conjunctivitis, and (5) acute trachoma. Table 1 illustrates the essential points of difference among these conditions. It will be observed that acute herpetic keratocon-

* From the Division of Ophthalmology, University of California Medical School. This work was made possible by funds donated by Mrs. Clara Heller. Presented at the 84th annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, May, 1948.

junctivitis offers the greatest difficulty. Its gross resemblance to epidemic keratoconjunctivitis is striking but differential diagnosis can be made readily on the basis of slit-lamp examination since the corneal lesions in the herpetic disease are invariably epithelial and those of epidemic keratoconjunctivitis invariably subepithelial.

TABLE 1

DIFFERENTIAL DIAGNOSIS OF EPIDEMIC KERATOCONJUNCTIVITIS

	Epidemic Kerato-conjunctivitis	Herpetic Kerato-conjunctivitis	Inclusion Conjunctivitis	Beal's Conjunctivitis	Nummular Keratitis	Acute Trachoma
Preauricular adenopathy	++++	++++	+	+	0	±
Follicular hypertrophy	++++	++++	++++	++++	0	++
Pseudomembranes	++	++	0	0	0	±
Keratitis	Subepithelial round infiltrates	Epithelial infiltrates; dendritic figure	0	0	Round subepithelial infiltrates; facet formation	Pannus; irregular infiltrates
Visual disturbance	++++	++++	0	0	Variable	Late
Cytology of conjunctival exudate	Mononuclear cell exudate	Mononuclear cell exudate	Polymorphonuclear cell exudate	Mononuclear cell exudate	Not known	Polymorphonuclear cell exudate

PERSONAL OBSERVATIONS ON EPIDEMIOLOGY

An attempt was made in every case observed to work out the source of infection. The fact that the incubation period is usually from 7 to 10 days helped in tracing possible contacts. The cases could be divided into five epidemiologic groups, as follows:

1. SOURCE OF INFECTION UNKNOWN

Patients in this group could not recall having been in contact with anyone with inflamed eyes, nor had they, within the period of incubation, been in attendance at any doctor's office where they might have been accidentally exposed. In view of the possibility that the infection may be carried in the respiratory tract, each patient was questioned as to the presence of respiratory disease in his family or among his immediate contacts, but the incidence was no greater among the epidemic keratoconjunctivitis cases than it was in a similar series of patients without the disease. It is noteworthy that the great majority of patients in this group were young, active adults who were in contact with numerous people in their work or in traveling to and from their work; there was not one who was not exposed repeatedly to persons other than those in his immediate family.

2. FINGER-TO-EYE TRANSMISSION

This type of transmission was presumed to be the cause of the infection of three ophthalmologists, each of whom contracted the disease after treating one or more typical cases of it.

Case 1. Ophthalmologist A had 17 cases in his practice and developed a fulminating case of epidemic keratoconjunctivitis with thick membranes. The character of the membranes was such as to suggest diphtheria, but repeated cultures failed to reveal pathogenic bacteria. It was not until the typical corneal picture of the disease developed that

a definite diagnosis was made. In handling his cases, this ophthalmologist had used the ordinary hand-washing technique. No solutions had been used in his eyes. His wife subsequently contracted a mild case of the disease.

Case 2. Ophthalmologist B had five cases of typical epidemic keratoconjunctivitis in his practice and developed a severe case of the disease shortly after going on vacation. He had employed the usual soap and water hand wash after treating his cases. No secondary cases developed in his family.

Case 3. Ophthalmologist C developed a severe case of epidemic keratoconjunctivitis after seeing only one case, the nature of which he had not recognized until corneal signs developed. He also had employed the usual soap and water hand-washing technique. No secondary cases developed in his family.

None of the three ophthalmologists recalls rubbing his eyes after seeing cases, but this is a very common gesture and often an unconscious one. Fingers-to-eyes is believed to have been the most likely route of infection.

In addition to these three ophthalmologists seen by me personally in the 1947-1948 epidemic, I know of eight other such cases and there must surely have been still others. With reference to the 1941-1942 epidemic, I was able to obtain from colleagues and from the literature the history of seven infections in ophthalmologists. Except for one doctor whose eyes were accidentally sprayed with irrigating fluid, all were apparently the result of finger-to-eye transmission.

3. Familial transmission

Three cases were observed in which it was clear that the husband had transmitted the disease to his wife. One of these transmissions was from one of the ophthalmologists mentioned above. The infection developed in his wife 17 days after the onset of his own disease. The wife had not treated her husband's disease in any way but she was in intimate contact with him during the period of his illness. The exact mode of transmission could not of course be determined, but of the articles handled by both, sheets and pillowcases would seem to have been the most likely agents of transmission in view of the abundant tearing characteristic of the infection.

The other two husband-to-wife transmissions were comparable. Both couples slept in double beds where contaminated linen would have offered a likely means of transmission.

4. Industrial infections

During 1947 and the first four months of 1948, I observed only one individual with the disease who was employed in the shipbuilding industry. There were no other cases in this man's shop and he is believed to have contracted the disease while being treated for chronic conjunctivitis in a doctor's office in which numerous typical cases of epidemic keratoconjunctivitis were being seen.

Other patients with the disease were engaged in the following occupations: canning, meat packing, plumbing, merchandising, agriculture, law, insurance, grocery store clerking, secretarial work, and nursing. Except in the case of the one nurse and one secretary, no history of contact with other cases in connection with the patient's work could be obtained. The nurse was a public health worker who was exposed to a number of individuals with inflamed eyes but she did not know that a diagnosis of epidemic keratoconjunctivitis had been made in any of them. The secretary was employed in an internist's office and was in contact with several patients infected with the disease.

In returns from questionnaires sent to representative opthalmologists throughout the country, there were no histories of outbreaks in any occupational group except the physician group. I was unable to elicit any history of the disease in welders or shipyard workers. This was, of course, sharply at variance with the situation in 1941-1942

when the disease was epidemic among shipyard workers, and in particular among welders.

5. Office infections

Transmission of epidemic keratoconjunctivitis in offices and industrial clinics has been noted repeatedly in the literature. Such accidental transmissions were not uncommon in the 1941-1942 epidemic and they occurred again with important frequency in the 1947-1948 outbreak. I have seen in consultation patients from four series of accidental office transmissions and have been informed of a number of other series. Three colleagues were kind enough to furnish details of office transmissions in which the mechanism of transmission could be worked out with reasonable reliability. These are reported as follows:

Office A. In Office A (table 2) there were 19 infections, all believed to have been derived from a single case. In the first 14 of these the only common factor was the administration of a 0.5-percent pontocaine solution from a dropper bottle. Tonometry and hand contamination could also have been factors in some of the transmissions. The majority of the 14 were glaucoma cases in which tonometry had been performed, but all 14 had received the pontocaine solution and some the pontocaine only. How long the pontocaine remained infective could not be determined as the solutions were discarded and the bottles sterilized at irregular but frequent intervals. Unfortunately there was no record of the day upon which this had been done. However, the epidemiologic data indicate that all infections occurred on one day, since the first 14 cases were seen on a single day and all developed first symptoms nine days later.

On the ninth day several of the 14 cases appeared in the office for treatment. When the first appeared, the probable source of his infection was surmised and the dropper bot-

TABLE 2

Cases of epidemic keratoconjunctivitis developing in offices A and B

	Office A		*Office B*	
Patients developing epidemic keratoconjunctivitis after receiving 0.5-percent pontocaine solution from dropper bottle	14		42	
Percentage of those exposed to solution who developed infection		90 to 100%?		98%
Patients developing infection who had *not* been exposed to contaminated solution	5		0	
Secondary cases arising from office infections	2		0	
Total number of cases	21		42	
Incubation period	9 days		7 to 10 days	
Patients developing				
(a) keratitis	21	100%	42	100%
(b) pseudomembranes	2	10	3	3
(c) preauricular adenopathy	21	100	36	85
(d) bilateral disease	18	90	37	90
(e) secondary infection	0	0	0	0
(f) dendritic keratitis or lid vesicles	1	5	0	0
Role of trauma	Tonometry, meibomian gland expression, removal of sutures, or other manipulation in all but one case		Tonometry in majority of cases	

tle technique of administering the pontocaine solution was promptly discarded. In spite of this precaution, five new cases developed after another 9-day interval. Neither pontocaine solution nor tonometry could have been the agents of transmission in any of these cases. The hands of the attending ophthalmologist could have been responsible, or possibly the arms of the treatment chairs could have been contaminated. One of the five cases did not enter a treatment room, however, but received euphthalmine drops for funduscopy in the darkroom where it is believed none of the infected patients had been.

Two additional cases were treated in this office at this time, both of them contracted secondarily by the spouses of 2 of the 19 office infections.

Office B. The accidental infections which occurred in Office B (table 2) numbered 42 and, unlike those in Office A, all cases had received 0.5-percent pontocaine solution from a single dropper bottle over a period of eight days. A good many cases had been subjected to tonometry but the only factors common to all were the use of pontocaine and examination in the treatment room. Since no patient examined in the treatment room who did not receive pontocaine developed the infection, it would seem reasonable to eliminate these other factors from consideration.

Unlike the situation in Office A, there were no known secondary infections from any of the office cases. The reported incubation period of 7 to 10 days is at variance with the sharp 9 days observed in Office A, but more careful scrutiny of the data on this point might yield closer agreement.

Office C. The infections which occurred in Office C furnished particularly valuable data on the epidemiology of the disease. The incubation period varied from 8 to 13 days in the 11 cases in which it was known, as follows: 8 days in 3 cases, 10 days in 3, 11 days in 3, 12 days in 1, and 13 days in 1. It is of interest that in those cases in which only one eye was treated in the office, only that eye initially developed the disease; whereas, in the patients who had had both eyes treated, bilateral infection occurred. This would seem to indicate a high degree of susceptibility to the virus. All the cases which developed in this office had preauricular adenopathy and keratitis. If the cocaine-adrenalin used in the first office patient with epidemic keratoconjunctivitis was contaminated with the virus, the duration of its infectivity must have been protracted, since the first office infection did not receive the drops until 11 days later and the last infection in the first series did not receive them until 41 days later.

The following paragraphs present the reconstructed history of the 3 small outbreaks of the disease which occurred in Office C:

Series 1 (1941)

Case 1. September 7th. Patient was treated for what appeared to be an acute catarrhal conjunctivitis, left eye. The eye was cocainized and conjunctival scraping taken and found to be negative. Patient was not seen again until October 19th when there were numerous infiltrates, left eye, typical of epidemic keratoconjunctivitis. It is probable that this infection had just developed when patient was first seen in early September.

Case 2. September 18th. Patient was refracted in the office. Eleven days later (October 1th), onset of typical epidemic keratoconjunctivitis with preauricular adenopathy and corneal infiltration, both eyes.

Case 3. September 24th. Foreign body removed from upper tarsal conjunctiva. Eight days later (October 2nd), onset of conjunctivitis; developed into typical epidemic keratoconjunctivitis.

Case 4. September 25th. Conjunctival concretions in the right eye were removed. Eight days later (October 3rd), onset of conjunctivitis, right eye; developed into typical epidemic keratoconjunctivitis, later spread to left eye.

Case 5. October 6th. Abrasion of con-

junctival follicles, left eye. October 14th (8 days later) conjunctivitis started in left eye, developed into typical epidemic keratoconjunctivitis.

Case 6. October 8th. Chalazion operation, right eye. Eleven days later (October 19th), typical epidemic keratoconjunctivitis developed.

Case 7. October 18th. Mild conjunctivitis, both eyes. October 29th (11 days later) epidemic keratoconjunctivitis developed in both eyes.

Between November 1st and January 1st there were four more cases, seemingly not connected with one another, but in this period there were no office infections.

Cases 3, 4, 5, and 6 received cocaine-adrenalin drops which could have been from the same source as those used on September 7th on Case 1. However, Cases 5 and 6 had minor surgery and Case 7 an examination for conjunctivitis on days when Cases 2, 3, and 4 also made office calls. Cocaine-adrenalin drops were used in Cases 1, 3, 4, 5, and 6, and in all 7 cases there was manipulation of the lids.

Series 2 (1917)

Case 1. June 17th to 21st. An eye physician with bilateral epidemic keratoconjunctivitis was seen in consultation in Office C.

Case 2. June 17th. Tear duct, right eye, irrigated because of complaint of tearing. First sight of conjunctivitis, right eye, June 26th. Typical epidemic keratoconjunctivitis developed.

Case 3. June 21st. Patient prepared for cataract operation: tear duct irrigated, tension taken, meibomian glands massaged. Cataract operation, June 22nd. Conjunctivitis started July 1st, developed into typical epidemic keratoconjunctivitis.

Case 4. June 21st. Exactly like Case 3.

All four cases received cocaine-adrenalin drops. Evidently Cases 2, 3, and 4 were office infections, from these drops or from contaminated hands. However, since the attending ophthalmologist had diagnosed Case 1 as epidemic keratoconjunctivitis, he was very careful about his hand washing technique and therefore considers the infectious agent to have been in the drops or on the nipple of the dropper bottle. It is his opinion that the virus probably stays viable for hours or days on the rubber nipple, in the drops, or on the hands.

Series 3 (1917)

Case 1. September 20th. Right eye became inflamed about 10 days after an eye examination by an ophthalmologist in Office X; vision became blurred around October 1st. Observed in Office C on October 19th when there were numerous subepithelial infiltrates, both eyes.

Case 2. October 26th. Friend of Case 1 consulted same ophthalmologist in Office X. First observed in Office C on November 7th when numerous corneal infiltrates had developed in both eyes.

Case 3. November 23rd. Patient examined in Office X. First observed in Office C on December 13th when there were a few superficial punctate infiltrates.

All three of these patients developed typical epidemic keratoconjunctivitis which in all probability they acquired in Office X between September 10th and November 23rd.

Epidemiologic control

The information regarding the epidemiology of epidemic keratoconjunctivitis is admittedly incomplete due to lack of laboratory controls. Until the incidence of neutralizing antibodies for the virus in the general population is known, little can be said concerning inapparent or subclinical infections. It is believed, however, that one attack confers a permanent immunity and it is noteworthy that it has been impossible to obtain a history of a second attack from any of my own cases or from the records of my colleagues. The data from the office infections reported above indicate that there is a high degree of susceptibility in the gen-

eral population and would suggest that subclinical infections are rare.

On the basis of the experience gained in the 1941-1942 epidemic, a number of recommendations for the prevention of the disease were promulgated[4-7] but unfortunately were not generally adopted. Of these recommendations, the following would seem to be the most important:

1. Not only ophthalmologists, but general physicians and nurses, particularly those connected with industrial dispensaries, should become thoroughly familiar with the clinical characteristics of the disease in order to facilitate early diagnosis.

2. Physicians and nurses should be meticulous in the washing of their hands between examinations. This washing should be with soap and water and could well include scrubbing with a brush.

3. Dropper bottles should be discarded from all eye offices and industrial dispensaries. Individual sterilizable droppers should be used routinely.

4. Office procedures should be modified so that epidemic keratoconjunctivitis cases can be seen apart from other cases. Protection against contamination of treatment chair arms and doorknobs should be instituted.

5. All instruments, including tonometers and contact lenses, which are used on patients should be sterilized.

6. Patients with epidemic keratoconjunctivitis should receive instruction in the use of tissues in eye care to prevent hand contamination. Separate sleeping arrangements should serve to diminish husband-and-wife infections.

7. Common use of masks and goggles in industry should be discouraged.

Discussion

The literature on epidemic keratoconjunctivitis reveals a considerable degree of confusion as to the relationship of the disease to superficial punctate keratitis, nummular keratitis, acute follicular conjunctivitis (Beal), and herpetic keratoconjunctivitis. The work of Sanders[3] has shown clearly that epidemic keratoconjunctivitis is a specific virus disease. Future laboratory studies will no doubt clarify the picture, but on clinical grounds alone a reasonable differentiation is possible at the present time.

Superficial punctate keratitis, as we are familiar with it in the United States, is an entirely different disease,[9] with minimal conjunctival symptoms and a chronic course. The punctate corneal lesions are not grossly visible like the round infiltrates of epidemic keratoconjunctivitis and are epithelial rather than subepithelial. It is true that epidemic keratoconjunctivitis was originally described by Fuchs[10] under the name "superficial punctate keratitis," but usage in this country has identified this name with a disease entity of which it is more closely descriptive.

The term "keratitis nummularis," although originally identified with the entity now known as epidemic keratoconjunctivitis, has since been used to describe a quite different disease[11] with insignificant conjunctival lesions compared with the corneal lesions, and with a tendency of many of the corneal infiltrates to form facets and even ulcers, a tendency entirely lacking in epidemic keratoconjunctivitis.

Acute follicular conjunctivitis (Beal) has, in my experience, never been complicated with keratitis. It has a shorter course than epidemic keratoconjunctivitis (never longer than three weeks), moreover, and has never been known to develop pseudomembranes or grossly visible preauricular adenopathy.

Herpetic keratoconjunctivitis is a rare disease in which the conjunctival signs exactly simulate those of epidemic keratoconjunctivitis but in which the corneal signs develop without the delay characteristic of epidemic keratoconjunctivitis and are epithelial rather than subepithelial. The occurrence of a dendritic figure is of course differentiating. The studies of Maumenee and his associates[12] suggest a relationship between

this herpetic disease and epidemic keratoconjunctivitis but their work has yet to be confirmed.

Epidemic keratoconjunctivitis is not the only infection in which transmission has occurred in offices and clinics. The role of the tonometer in the transmission of inclusion conjunctivitis has been described (Thygeson and Stone[13]) and the role of the contaminated solution bottle in the production of pyocyaneus ulcers has been well established (McCulloch[14]). No other infection has produced such widespread office outbreaks, however, and no other disease has produced a comparable number of infections in doctors and nurses. It is obvious that a thorough reëxamination of office and dispensary techniques is indicated.[6]

The mechanism of spread in office outbreaks is not difficult to work out but the epidemiology of sporadic cases is obscure. Does the lack of known contact with typical cases indicate that subclinical disease is a factor? Elucidation of this question must await further laboratory studies on the general population. On purely clinical grounds, however, it seems within the realm of the possible that a single case of epidemic keratoconjunctivitis could contaminate enough doorknobs, toilet levers, washbasin faucet handles, drinking fountain handles, and so on, to account for sporadic cases of the disease over a wide territory.

Summary and Conclusions

1. Epidemic keratonconjunctivitis, which first appeared in the western United States in epidemic form in the fall of 1941, became widespread throughout the country in 1942. During the war years of 1943-1946 only sporadic cases were seen and, contrary to expectation, the disease did not become a military problem. In 1947, the disease again became epidemic in California and, to a lesser extent, in other parts of the country.

2. The American disease is believed to be identical with the keratoconjunctivitis first described in Vienna, in 1889, under different names by Adler, Fuchs, and others. It should be differentiated from acute herpetic keratoconjunctivitis, nummular keratitis (Dimmer), acute follicular conjunctivitis (Beal), acute trachoma, and so on. It bears no relationship to superficial punctate keratitis, an entity which has been widespread in this country.

3. The high communicability of the disease appears to be due to the ability of the virus to survive drying and dilution. In this respect it differs from other potentially epidemic types of keratoconjunctivitis such as gonorrheal ophthalmia and trachoma.

4. In the present study, transmission of the disease has been shown to have occurred by the following means: (a) contaminated tonometers, (b) contaminated solutions, including pontocaine, cocaine-adrenalin, and homatropine, (c) direct finger-to-eye transmission, and (d) fomites, especially welders' masks, goggles, and common tools.

5. No satisfactory evidence was obtained to indicate the presence of asymptomatic conjunctival carriers or respiratory carriers.

6. In view of the high frequency of office transmissions, routine office practice should be reëxamined in order to prevent this and other infections. The following prophylactic measures are to be recommended: (1) The discarding of all dropper bottles, (2) the use of individual sterilizable droppers, (3) adequate hand washing before and after treatments, (4) the use of disposable treatment chair arm covers, and (5) individual masks and goggles for workers in industry, with early recognition and isolation of cases.

87 North 6th Street.

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ACCOMMODATIVE DEFECT FOLLOWING ATMOSPHERIC CONCUSSION*

HOMER E. SMITH,† M.D.
Salt Lake City, Utah

The effects of atmospheric concussion on the ocular structures have long been recognized. Stoewer,[1] as cited by Wurdemann,[2] described a multiplicity of ocular pathologic findings resulting from concussion following the terrific explosion at the Roburit factory long before World War I. During and following World War I, Lagrange,[3] McKee,[4] Tooke,[5, 6] deSchweinitz,[7] Doherty,[8] Anderson,[9] Lister,[10] Slight and Haughey,[11] and others[12, 13] rather extensively covered the subject matter pertaining to ocular lesions resulting from atmospheric concussion in military and civilian casualties seen by them during that war.

The effects of atmospheric concussion on the eye during World War II have been described by Stallard,[14] Mathews,[15] Tyrrell,[16] and others.[17-29] However, in spite of the extensive writings to date, manifestations of doubt as to the efficiency of atmospheric concussion (windage) in producing ocular damage appear in the literature.[28-29]

The studies of Theis,[30] Sutherland,[31] and others,[32-35] have shown the essential damaging physical qualities of atmospheric blast to be: (1) An excessively intense longitudinal compression wave of short duration travelling at a velocity of 5,000 to 25,000 feet per second, with a pressure component up to 200 atmospheres (3,000 pounds) per square inch, followed by (2) a phase of rarefaction (suction component) which is never less than 15 pounds per square nich (perfect vacuum). Both components are capable of producing injury.

It is natural that the eye should share, with other organs, the damage sustained by concussion. In the absence of gross lesions, minor changes may be found in the ocular structures which are of value to the ophthalmologist in his clinical evaluation of patients who fall into this postconcussion class.

Many articles have been presented recently[36-45] describing the cerebral and mental effects following atmospheric or blunt traumatic concussion, and their manifestation following such exposure has become known as the "postconcussion state."[44] The essential symptoms that have been described in this syndrome are: headache, dizziness, fatigability, impairment of memory, poor concentration, sensitivity to temperature change, character changes, emotional instability, and antisocial behavior.

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† From the Department of Surgery, Section of Ophthalmology, University of Utah Medical School.

Among service personnel (the source of this material) other motivating factors were effective in precipitating similar emotional and personality disturbances. To rule out these in patients who had a history of exposure to atmospheric concussion, a search was made for objective evidence of ocular pathologic conditions which might have resulted from such trauma.

While I was on active duty in the U.S. Navy Medical Corps, many such postconcussion cases were observed in which relatively minor pathologic ocular findings appeared to substantiate a history of exposure to atmospheric concussion of traumatizing degrees. The patients had been hospitalized on the neuropsychiatric service with the complaints of irritability, restlessness, inability to concentrate, lassitude, periodic emotional swings, memory deficiency, headaches, rapid ocular fatigue and pain upon reading, blurred vision, ocular pains, and ear symptoms of tinnitus and pain. They were referred to the department of ophthalmology by the neuropsychiatric service for evaluation of their eye complaints. These cases presented evidence of mild but definite ocular changes in comparison with those seen in severe concussion injuries. Although minor in degree of severity, they were uniformly consistent and of such constancy as to offer definite aid in their analysis for the neuropsychiatric service.

Case reports

Four such cases are presented and, in Case 1, a 20-month follow-up examination was made.

Case 1

History. A 35-year-old lieutenant was admitted to the hospital on October 10, 1943, with the complaints of diminished hearing, numbness, marked fatigue, stuttering, memory defect, mental lethargy, emotional instability, insomnia, speech difficulty, occipital headaches, blurred distant and near vision, poor night vision, and bilateral eye pains since exposure to a severe bomb blast on October 1, 1943.

The physical examination was essentially negative except for the significant aural, neuropsychiatric, and ocular findings. His hearing was reduced and bone conduction exceeded air conduction.

The neurologic findings revealed an increase in the deep tendon reflexes on the left, a questionable positive Babinski on the right, some impairment of coördination in the use of the upper extremity muscles, mental slowness, confusion in executing instructions, and hesitancy in selecting words for speech. It was the opinion of the neurologist that there was present a diffuse organic condition simulating the picture of multiple sclerosis which could have been due to multiple areas of edema or hemorrhage resulting from the concussive effects of the explosion.

The psychiatric consultant reported, "In my opinion this man shows a very appreciable personality change of the type associated with concussion. These changes are regressing, but I am certain a Rorschach test, even at the present time, would reveal an organic pattern. I believe this man is unfit for combat duty, and should be under prolonged neuropsychiatric observation because of the possibility of (1) marked personality changes and (2) convulsive episode."

The eye findings revealed a mild degree of episcleral injection near the limbus of each eye. Vision was: O.D., 20/25; O.S., 20/30. With a pinhole disc the vision of each eye was reduced to 20/40. Unfortunately, accommodation measurements were not taken. The homatropine cycloplegic refraction revealed: O.D., with a +0.75D. sph. ⌒ +0.5D. cyl. ax. 80° = 20/20; O.S., with a +0.75D. sph. ⌒ +0.25D. cyl. ax. 100° = 20/30.

The ophthalmoscopic examination revealed hazy media, pigment clumps on the anterior lens capsule, and fine products of inflammation in the vitreous. The discs were slightly elevated, the margins blurred, and the central excavation was filled with white edematous tissue. The retinas showed diffuse edema throughout the whole of the fundi, being especially marked in the equatorial and peripheral regions. In these regions there were also areas of mild exudative choroiditis. In the right fundus, one disc diameter temporal and down from the macula there was a one-fourth torsion of the edematous retina and its vessels with a small hole. The maculas showed fine edema, distortion of the foveal reflex, and mild retinal pigment epithelium proliferation.

The visual fields revealed a peripheral constriction to a 2-mm. white target at 330-mm. distance which varied from 15 to 25 degrees in extent (fig. 1). X-ray films of the skull were negative.

Course. Three weeks later the patient was transferred to another hospital and was returned to full duty on December 31, 1943.

Follow-up study. On August 14, 1945, the patient was again admitted to a naval hospital with the chief symptoms of emotional swings, mood changes, lack of interest, and personality changes. The physical examination was essentially negative.

Eye findings. Corrected vision was 20/20 in each eye with the homatropine cycloplegic refraction findings of: O.D., +0.25D. sph. ⌒ +0.75D. cyl. ax. 75° = 20/20; O.S., −0.25D. sph. ⌒ +1.0D.

cyl. ax. 95° = 20/20. The amplitude of accommodation was: R.E., 3.3 diopters (30 cm.); L.E., 5.9 diopters (17 cm.). The peripheral and central fields to a 3-mm. white target were normal. A slight ptosis of the right upper lid existed, and the right pupil responded more to cocaine pupillary studies than the left. Versions and phorias were within normal limits.

The fundi revealed the discs to be round, flat, with distinct margins and tissue proliferation in the physiologic cups. The vascular structure appeared to be normal. The maculas showed fine pigmentary changes, distorted foveal reflexes, and

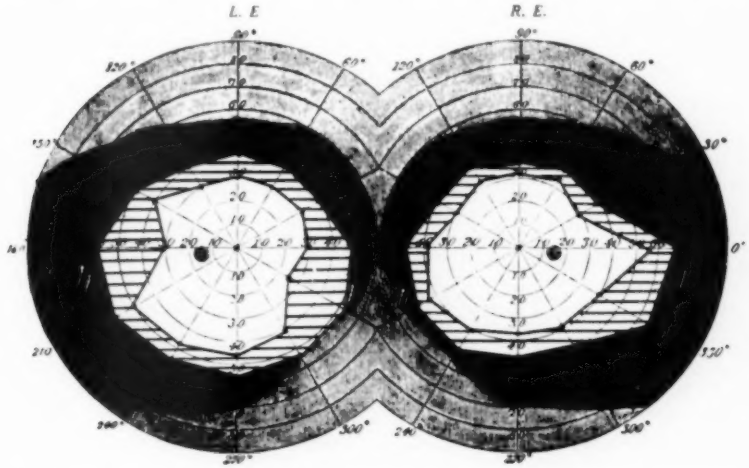

Fig. 1 (Smith). *Case 1.* Visual fields, taken on November 2, 1943, revealed a peripheral constriction to 2/330 white.

reflexes radiating out from their center like spokes in a wheel.

The retina in the right eye revealed a small proliferating retinal scar lying between two anomalously directed blood vessels that were incorporated in the "whorl" of the previous findings. From this scar, radiated additional spokelike reflexes.

In the equatorial region and periphery there were diffuse omnipresent fine retinal pigmentary changes of dispersion and proliferation. Associated with them were definite, fine changes in the retina which appeared to be cystic in nature and which rendered the tissue somewhat translucent.

The laboratory findings were essentially normal. The spinal fluid showed: W.B.C., 2; R.B.C., 3; sugar, 62 mg. per 100 c.c.; protein, 30.2 mg.; chlorides, 790 mg.; globulin, faint trace; negative Kahn; colloidal gold curve, 1,123,210,000. An electroencephalogram tracing was within normal limits.

CASE 2

History. This 23-year old white, Marine combat man was admitted to the hospital on July 5, 1945, with the complaints of frontal headaches, chest pains, antisocial attitude, restlessness, nightmares, inability to concentrate, blurred vision on detailed use of the eyes, and earache.

The past history (medical records substantiated the patient's story) revealed that on February 19, 1945, while on Iwo Jima, the patient was knocked unconscious for an indefinite period as a result of blast concussion (mortar-shell explosion). Upon awakening, the patient was unable to hear or speak and noticed great difficulty in reading. The hearing began to improve in 24 hours, but the speech defect persisted for 6 days. The record states that there was no evidence of marked emotional disturbance.

The family history was essentially negative.

The physical examination revealed the following significant findings. There was a 40 percent hearing loss in the left ear and Shrapnell's membrane was red on the left side.

Eye findings. Vision was: O.D., 20/20; O.S., 20/25, and the amplitude of accommodation was 2.4 diopters (42.3 cm.) in the right eye and 2.3 diopters (44 cm.) in the left eye.

The cycloplegic (homatropine hydrobromide) refraction was: O.D., +0.75D. sph. ⊃ +0.25D. cyl. ax. 95° = 20/20; O.S., +0.5D. sph. ⊃ +0.37D. cyl. ax. 80° = 20/20. Versions and phorias were within normal limits. His PcB. was 25.5 cm. Perimetric studies were normal. The external ocular examination was essentially normal and slitlamp findings were within normal limits.

The fundi revealed the media to be clear, the discs were round, flat, and had distinct margins. There was increased visibility of the fine vessels. The appearance of the retinas, as a whole, was

that of semitranslucence from edema. In the equatorial regions and periphery, the edema increased in degree to produce moth-eatenlike areas of gray-white retinitis, irregular in shape and size, which were deep in the retina as determined by parallax. Diffuse, small, retinal pigment clumps were present. In the choroid, evidence of mild healed choroiditis was present and, in the left eye, a disc-sized hemorrhage was present in the peripheral choroid below. The maculas appeared to be normal.

The laboratory findings were essentially negative.

The neuropsychiatric consultation revealed no neurologic disturbance. The psychiatric evaluation revealed, "a coöperative but tense individual with normal speech. Combat rumination is interfering with thought processes. Affect is appropriate and grasp good. Service history confirms a severe blast concussion syndrome in February, 1945, on Iwo Jima with neuropsychiatric disability at that time. His present eye and somatic pain plus thought irregularity, are considered due, in the main, to his recent organic brain injury. Unfit for service of indefinite duration and recommend that he be discharged from the U.S. Naval Service."

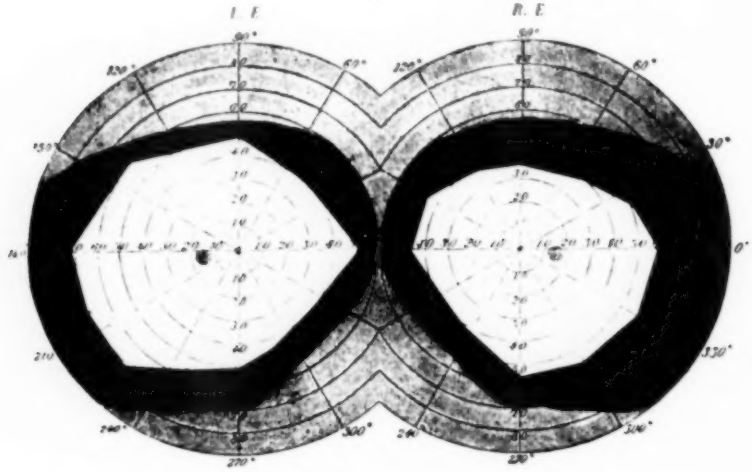

Fig. 2 (Smith). *Case 3.* Visual fields, taken on September 19, 1945, showed a 15-degree constriction to 3/330 white.

Case 3

History. The patient, a white man, aged 20 years, was a private first class in the U.S. Marine Corps, who was admitted to the hospital on September 17, 1945, with the complaints of a shaky feeling, headaches, rapid ocular fatigue when reading, general fatigability, and restlessness during sleep. These symptoms had begun 7 months previously, following exposure to the concussion of a mortar-shell explosion 10 feet from him. The patient stated that he was unconscious for 12 hours following the trauma. There had been gradual improvement in his condition since then.

The authenticated past history revealed that the patient was first admitted to the sick list with the diagnosis of "blast concussion, atmospheric," on February 19, 1945, with the symptoms as he described. He continued as a patient in the naval medical facilities until May 23, 1945, at which time he was discharged to limited duty. The family history was essentially normal, as was the general physical examination.

Eye findings. Vision was: O.D., 20/20; O.S., 20/20. Cycloplegic (homatropine hydrobromide) refraction revealed: O.D., +0.75D. sph. ⌒ +0.25D. cyl. ax. 100° = 20/20; O.S., +0.75D. sph. ⌒ +0.37D. cyl. ax. 80° = 20/20. The amplitude of accommodation was: R.E., 7.7 diopters (13 cm.); L.E., 6.9 diopters (14.5 cm.).

The fundi presented a slight but definite appearance of retinal edema in the periphery with the findings in the right eye being the most marked. Fine pigment changes were present in the pigment epithelium. Versions and phorias were within normal limits. Peripheral field (fig. 2) studies with a 3-mm. white target revealed a 15-degree constriction from normal.

The neurologic examination was negative and the psychiatric examination revealed mild mental tension, anxiety, and sleep disturbance. The patient was continued on limited active duty.

Case 4

History. This 24-year-old Marine corps ser-

geant (white) was admitted to the hospital on November 5, 1945, for reëvaluation of his symptoms of restlessness, irritability, and nightmares.

His authenticated past history revealed that on November 15, 1944, he suffered trauma to both eyes and head from "blast concussion, atmospheric," as a result of a hand grenade exploding directly in front of him. This incident was followed by tinnitus, ocular pain, and headache. In January, 1945, the patient began to show mental symptoms of nervousness, restlessness, irritability, and sleeplessness. In February, 1945, he was admitted to the sick list because of accentuation of these symptoms and was ultimately placed on limited active duty in the United States on April 18, 1945, with a diagnosis of "fatigue, operational." The physical examination at this time was essentially normal.

Eye findings. Vision was: O.D., 20/20; O.S., 20/20. Cycloplegic (homatropine hydrobromide) refraction showed: O.D., +0.75D. sph. = 20/20; O.S., +0.75D. sph. ⌒ +0.12D. cyl. ax. 90° = 20/20. The amplitude of accommodation was: R.E., 7.4 diopters (13.5 cm.); L.E., 7.2 diopters (13.8 cm.). The PcB. was 8.5 cm.

The external ocular examination and pupillary findings were within normal limits. The fundi revealed a diffuse pre-equatorial irregularity in the pigment epithelium. There was a translucent appearance to the retina in the periphery with areas of cystic degeneration. Occasional areas of increased and dispersed choroidal pigment were seen between the choroidal vessels which were interpreted as residue of previous choroiditis. The field studies with a 2-mm. white target showed a slight peripheral field constriction (fig. 3).

The neuropsychiatric examination revealed residual evidence of his occupational fatigue and it was felt that he was not qualified for full duty (fig. 4).

Discussion

The major damage sustained by the eyes as a result of exposure to atmospheric concussion has been elaborated upon in the past; however, the effect upon the ocular tissues of relatively minor degrees of atmospheric concussion producing lesser damage has been infrequently mentioned. From the cases cited above, it appears as if comparatively minor but important changes may occur in the tissues of the eye as a result of exposure to atmospheric concussion.

The changes seen in the fundi and the deficiency manifested by the accommodative apparatus were rather constant, and the latter was the apparent source of a constant symptom on the part of these patients—that is, fatigability upon reading or with close detailed work. These ocular lesions aided the neuropsychiatric department in evaluating the concussion trauma background which contributed to the psychiatric complaints of

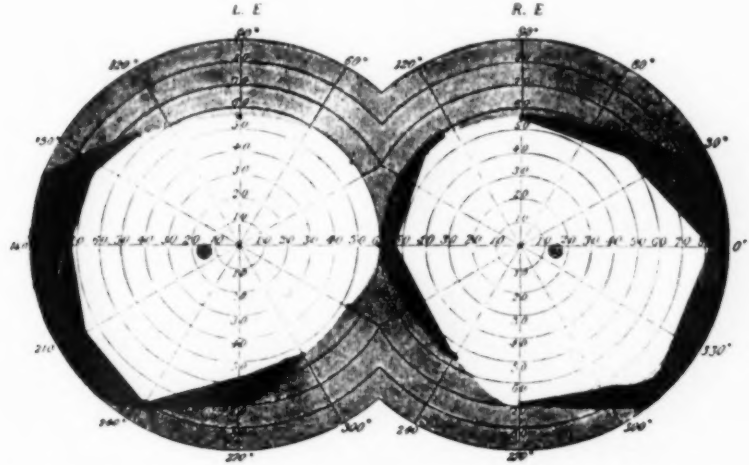

Fig. 3 (Smith). *Case 4.* Visual fields, taken on March 22, 1946, showed a slight peripheral constriction to 2/330 white.

patients who had been exposed to atmospheric concussion.

Westcott[46] and Groves[47] differ somewhat in their opinoin as to the disturbance to the accommodative power of the eye resulting from blunt traumatic concussion. The more extensive work of Wescott indicates that there is no significant posttraumatic change in the accommodative amplitude; rather, the reading discomfort experienced by these patients is due to a disturbance in the higher cortical centers.

In our cases the inability to read with comfort seemed to be directly attributable to the defect in the accommodative apparatus. The pathologic defect in the accommodative mechanism is apparently in the ciliary body, which is consistent with the fundus changes increasing in degree and extent as the ora serrata is approached.

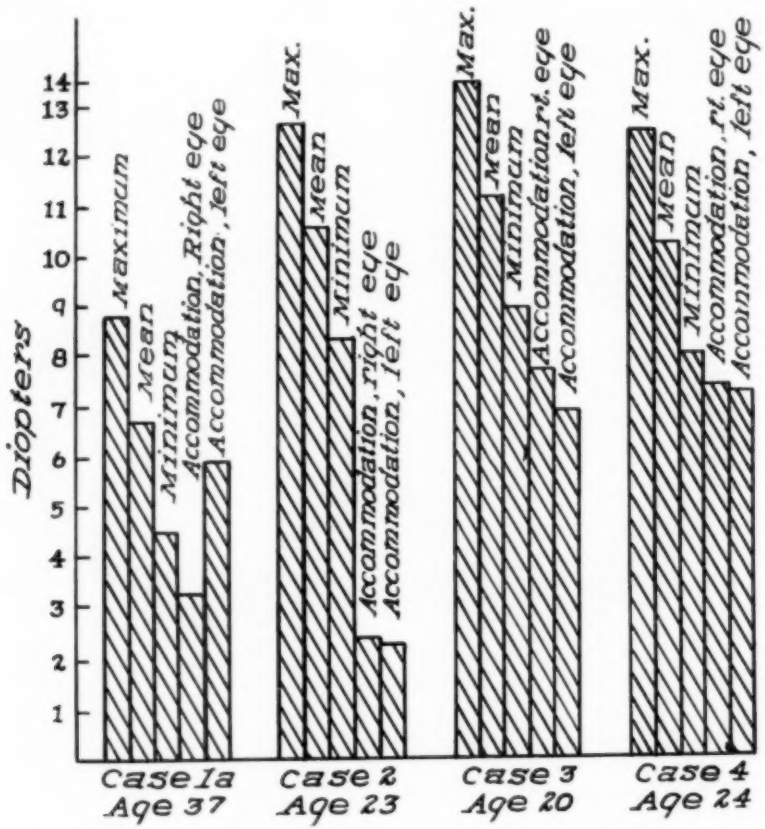

Fig. 4 (Smith). Accepted limits of accommodative values for patient's age (see reference 48) compared with the accommodative findings exhibited by the patients in this study.

Summary

Four cases with authenticated histories of exposure to atmospheric concussion of undetermined degree have been presented. They had been examined at posttraumatic intervals which varied in time from 10 days

to 12 months. In one case follow-up study was made after 20 months. All cases were seen as consultations for the neuropsychiatric service for evaluation of their ocular complaints.

The cases presented the following fundamental pathologic features:

1. Symptoms of headache, irritability, inability to concentrate, restlessness, fatigability, ocular fatigue and pain upon reading.
2. Deficiency in the amplitude of accommodation.
3. Retinal edema which was more marked in the equatorial and pre-equatorial regions of the fundus.
4. Mild peripheral choroiditis lesions.
5. Cystic degeneration of the periphery of the retina.
6. Constriction of the peripheral visual fields.

Conclusion

Atmospheric concussion may affect the ocular structures to a variable degree. In minor degrees, there appear disturbances in the ocular structure and function which are uniformly constant. These are of aid to the ophthalmologist in evaluating the ocular complaints, and help the psychiatrist in his evaluation of the part played by the trauma in patients who manifest the symptoms of the "postconcussion syndrome."

Furthermore, there appears to be no correlation between the eye findings in the atmospheric and blunt traumatic postconcussion cases.

Atmospheric concussion may produce an ocular syndrome characterized by deficiency of accommodation, retinal edema, mild choroiditis, and peripheral field constriction.

1005 Medical Arts Building (1).

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OPHTHALMIC MINIATURE

The examination of eyes affected with fungus haematodes, in its early period, has led to the conclusion that the disease consists in organic change of the retina, or in a growth from the retinal extremity of the optic nerve. This would account satisfactorily for the appearance, exhibited in the commencement, of an adventitious growth in the fundus of the eye.

Sir William Lawrence, *A Treatise on the Diseases of the Eye*, 1833.

VERTICAL NYSTAGMUS ON DIRECT FORWARD GAZE WITH VERTICAL OSCILLOPSIA*

MORRIS B. BENDER, M.D., AND WARREN F. GORMAN, M.D.
New York

Vertical nystagmus may be present either on upward gaze or on direct forward gaze. Vertical nystagmus on upward gaze is not uncommon. As a rule, it is indicative of acquired disease of the brain stem.[1] Vertical nystagmus on direct forward gaze, however, is rare and is usually found in a patient with "congenital" nystagmus.†

On rare occasions, vertical nystagmus on direct forward gaze may be seen in patients with acquired disease of the nervous system. In these cases of vertical nystagmus it is necessary to distinguish the congenital from the acquired type. Although the differential diagnosis depends on a consideration of all the factors in each case, the presence of oscillopsia is frequently a deciding feature.

Oscillopsia is a visual experience in which the patient sees a stationary object as moving from side to side (horizontal oscillopsia) or up and down (vertical oscillopsia). The oscillopsia is concomitant with the rhythmic movements (nystagmus) of the eyes and it is usually indicative of an acquired nystagmus. The following two case reports are studies of patients with acquired upward nystagmus on direct forward gaze.

REPORT OF CASES

CASE 1

History. E. M., a 55-year-old man, was admitted on January 2, 1948, to the Bellevue Psychiatric Hospital. For two days before admission he felt weak and had the sensation of "falling backwards." The past medical history disclosed that in 1945 he was rendered momentarily unconscious by a blow on the head. In 1942, he had a discharge from the right ear, with subsequent tinnitus and poor hearing. He later admitted to consuming a quart of sherry wine daily.

On examination he was found to be emaciated, with dry peeling skin, fissures at the corner of the mouth, and injection of the conjunctivas with vascularization about the limbus of both eyes. There was frequent blinking, avoidance of light, and other signs of photophobia. (The patient claimed that he had always been sensitive to light.)

There was a constant tremor of the entire body, and a coarser tremor of the right lower extremity. He walked slowly on a broad base, falling to the left or backward. Both ankle jerks were diminished. There was plantar hyperesthesia and calf tenderness. Vibratory sense was diminished in both lower extremities. All laboratory studies, including spinal fluid examination, X-ray studies of the skull and chest, gastro-intestinal series, liver chemistries, blood bromides, and blood Wassermann tests, were negative.

Ocular status. There was photophobia with frequent blinking and narrowing of the palpebral fissures. Eye movements were performed well in all directions of gaze. However, when he looked directly forward, there was a very rapid nystagmus in the vertical plane. The nystagmus took place entirely above the horizontal meridian and the fast component was in an upward direction.

As the patient looked to each side, the spontaneous vertical nystagmus became oblique, inclining about 45 degrees to either side. When the patient lay on his side or

* From the Department of Neurology, New York University College of Medicine, and the Neurological Service, Bellevue Psychiatric Hospital. This work was aided in part by a grant from The Dazian Foundation.

† In the congenital form, vertical nystagmus on forward gaze is not the only ocular finding. It is frequently associated with a nystagmus on lateral, upward, and downward gaze. Often in these cases there is a history that the patient has had "dancing eyes" for many years.

back, the nystagmus continued unchanged relative to the long axis of the body. Closing one eye or both eyes did not alter the nystagmus. On extreme upward gaze, however, the nystagmus increased. On downward gaze the nystagmus diminished. The nystagmus fluctuated in rate and amplitude. In time, it gradually diminished.

Subjectively, the patient reported that objects "moved up and down"—vertical oscillopsia. He said that near objects oscillated less than far objects and not as rapidly.

Special Studies

A. Vestibular Tests

1. Turning tests. The patient was placed in a Bárány chair in a sitting position, with his head inclined 30 degrees forward. With his eyes closed, he was rotated 10 times in 20 seconds to either side. In a normal individual this procedure produces horizontal nystagmus in a direction opposite to that of rotation. In this patient the Bárány test modified the spontaneous upward nystagmus, resulting in an oblique (up and out) nystagmus.

The patient's head was then inclined 90 degrees toward the right shoulder and he was again rotated to the right and left. With the head in this position, rotation of normal people to the right (clockwise) produces downward nystagmus, and rotation to the left (counter-clockwise) produces upward nystagmus. On rotating the patient in this manner to the right, it was found that the expected downward nystagmus was not present. Instead, his spontaneous upward nystagmus ceased for 15 seconds, then slowly reappeared. On rotation to the left, the preëxistent upward nystagmus was increased in amplitude for 24 seconds, after which time the resting upward nystagmus became apparent at its usual amplitude.

2. Caloric tests. Aural examination revealed that there was no significant hearing loss. Each ear was irrigated for 60 seconds with ice water while the patient was sitting erect and his eyes fixating forward. Such irrigation of the right ear produced nystagmus to the left and irrigation of the left ear produced nystagmus to the right. There were no abnormal changes on the past pointing. Vertigo was present.

B. Optokinetic Nystagmus

A drum, 9 inches in diameter, papered with one-half inch alternate black and white vertical stripes, was rotated in front of the patient's eyes at a speed of 2 revolutions per second. In normal individuals, rotation of the drum with the axis in the vertical plane results in horizontal nystagmus. In our patient such rotation produced oblique nystagmus. With the axis of the drum in the horizontal plane, downward rotation accentuated, while upward rotation diminished the spontaneous upward nystagmus.

C. Effects of Drugs.

It is known that barbiturates influence nystagmus and other ocular movements.[2] Latent nystagmus or any other type of nystagmus which is present on forward gaze is abolished by intravenous injections of sodium amytal. In a previous communication[3] it was also reported that an ocular squint disappeared whenever that patient was intoxicated with alcohol. Evidently alcohol had the same effect as barbiturates. Consequently we tried the effect of the drugs (amytal and alcohol) in our two cases of vertical nystagmus on forward gaze.

1. Sodium amytal. Sodium amytal (0.2 gm.) was injected intravenously over a period of two minutes. Within two minutes the spontaneous upward nystagmus on forward gaze stopped and the oscillopsia disappeared. Deviation of the eyes in the vertical or horizontal plane resulted in nystagmoid movements but on many occasions the nystagmoid movements were fine and rapid.[4] During the period when the nystagmus on forward gaze was abolished by amytal, there was likewise no response to the optokinetic drum on forward fixation. We did not carry out Bárány-turning or caloric-stimulation

tests while the patient was under the influence of amytal. After 11 minutes, the spontaneous upward nystagmus on forward gaze and the concomitant oscillopsia began to return.

2. Alcohol. The patient was given 1,000 cc. of medicinal sherry in 10 minutes by stomach tube, after a gastric analysis. He became intoxicated and showed slurred speech, cheerful affect, and an increase in staggering. His body and extremity tremor diminished. The nystagmus on forward gaze ceased completely, as did the oscillopsia. However, as in amytal narcosis, nystagmus on lateral or vertical gaze persisted.

3. Hyoscine. It was suggested that the spontaneous nystagmus on forward gaze might be an extrapyramidal disorder. For that reason the effect of hyoscine on ocular movements was tried. Hyoscine (1/100 gr.) was injected subcutaneously. After 35 minutes, the patient complained of a dry mouth, and showed dilated pupils and a diminution of the tremor of the extremities. However, there was no effect on the nystagmus.

Course. The patient was given large doses of water-soluble vitamins orally and liver extract intramuscularly. The nystagmus fluctuated in rate and amplitude from day to day, but gradually subsided. Although, originally, lateral deviation of the eyes had resulted in oblique nystagmus, after 9 weeks lateral gaze produced largely horizontal nystagmus. The spontaneous vertical nystagmus above the horizontal meridian on forward gaze gradually diminished in rate and amplitude, being almost imperceptible 13 weeks after admission to the hospital. The peripheral neuropathy improved gradually.

Comment. This patient, a known alcoholic, showed vertical nystagmus and vertical oscillopsia on forward gaze. Under observation the nystagmus and the oscillopsia subsided, thus suggesting that the ocular signs and symptoms were of the acquired type. Of interest is that amytal given intravenously and alcohol by mouth temporarily abolished the vertical nystagmus on forward gaze and the associated oscillopsia.

Case 2

History. W. T. M., a 48-year-old white carnival operator, was admitted on October 8, 1947, to the Bellevue Psychiatric Hospital with early delirium tremens. On October 2, 1947, he noted that objects "jumped up and down"—(oscillospia)—"that the room seemed to turn around" and that he "saw double both to one side or above." He complained of dizziness and blurred vision. He also volunteered that "when one doctor examined me down here I couldn't turn my eyes to the left at all," thus suggesting a paralysis of conjugate gaze to the left.

Routine examination revealed signs of diffuse involvement of the entire nervous system. Defects in recent memory were present. There was a coarse irregular jerky tremor of all extremities. There were signs of peripheral neuropathy with absent knee and ankle jerks, diminished vibration sense below the knees, hyperesthesia to pin prick in the glove and stocking areas, calf tenderness and hyperpathia on stroking of the plantar surface of the feet.

All laboratory examinations, including spinal fluid studies, X-ray studies of the chest and skull, electroencephalogram, blood count, urinalysis, blood bromides, blood and spinal fluid Wassermann tests, and liver chemistry tests, were within normal limits. Gastric analysis showed that there was no free acid. It should be noted that the patient had had a subtotal gastrectomy for peptic ulcer three months before the present admission.

Ocular status. The patient was transferred to the neurologic service on October 10, 1947. He was observed daily during his hospital stay and at frequent intervals thereafter for a total of 9 months. Special eye examinations were periodically performed. It was noted that when the patient looked straight ahead the eyes moved rhythmically up and down (vertical nystagmus). The excursions

occurred entirely above the horizontal meridian and the quick component was in an upward direction. The eyelids moved in synchrony with the upward nystagmus. Position of the head did not alter this nystagmus. Subsequent examinations showed that with the head erect, lateral deviation of the eyes was also accompanied by an upward nystagmus but frequently there were rotary components.

On forward gaze there was also spontaneous zig-zag rotary movement of each eye, more so on the left. These zig-zag movements in one eye were neither coördinated nor associated with the movements in the other. Similar, almost myoclonic, movements were noted in various muscles throughout the body, especially in the tremulous upper extremities. On extreme downward gaze, the vertical nystagmus stopped. On moderate elevation of gaze, the vertical nystagmus became more conspicuous but, on extreme upward gaze, the nystagmus disappeared.

Fixation of near and far objects did not alter the nystagmus on forward gaze but the oscillopsia was much more apparent to the patient when he fixed objects at a distance. There was a latent weakness of both external rectus muscles. On occasions, when he attempted to look to the left, the right eye rotated to the left, but the left eye turned inward toward the nose. The mirror image of that pattern of movement took place on his attempt at right lateral gaze.

Convergence was good. The visual acuity on admission to the neurologic service was 20/100 and within a few weeks improved to 20/30 in each eye. Examinations of the central and peripheral fields of vision for form and color were normal.

Special Studies

A. Vestibular Tests

As already noted, the patient reported that the vertical oscillation of objects diminished as they approached him. Tests with targets at near and distant points revealed that the vertical oscillopsia disappeared when he fixed a target within one meter of his eyes. Beyond this point the vertical oscillopsia was pronounced. Bárány turning tests induced no significant abnormalities. Caloric tests, using cold-water irrigation of the ears, also yielded apparently normal reactions.

B. Optokinetic Nystagmus

On rotating a striped drum before the patient's eyes, the spontaneous nystagmus was altered. When the axis of the drum was horizontal and the rotation toward the patient,* the amplitude of the upward nystagmus was increased. When the direction of rotation of the drum was reversed, the eyes dipped below the horizontal axis and the upward nystagmus diminished in amplitude. On rotation of the drum in the vertical axis before the patient's eyes, clockwise or counter-clockwise rotation produced oblique (up and out) modification of the spontaneous vertical nystagmus.

C. Effect of Drugs

1. Sodium amytal. About two minutes after intravenous injection of 0.25 gm. of sodium amytal, the nystagmus on forward gaze began to decrease in amplitude. The vertical nystagmus on direct forward gaze disappeared completely 12 minutes after the injection was begun and remained absent for a period of 13 minutes. The oscillopsia likewise disappeared during this period. The zig-zag and completely dissociated eye movements were also abolished by amytal.

2. Alcohol. Intravenous injection of 80 cc. of ethyl alcohol, equivalent to 5 ounces of whiskey, caused the vertical nystagmus on forward gaze to disappear. The nystagmus on upward and lateral gaze, however, became more marked in amplitude. There were oblique components on lateral gaze. By the time tests with alcohol were made, the zig-zag eye movements were no longer present.

* In the normal individual such rotation of the drum produces slow deviation of the eyes above the horizontal axis with upward nystagmus.

Therefore, we have no information as to the effect of alcohol on these movements.

Course. Under observation the vertical nystagmus on forward gaze subsided and ultimately disappeared after 8 weeks. There was a concomitant disappearance of the vertical oscillopsia on forward gaze. Vertical nystagmus on forward gaze persisted for 12 weeks. Horizontal nystagmus on lateral gaze was still present after 5 months. Seven months after admission, there was slight rotatory nystagmus on left lateral gaze.

The peripheral neuropathy improved gradually. The knee jerks reappeared in 16 days, while the ankle jerks could be elicited again after 3 weeks. Calf tenderness disappeared after 9 weeks of hospitalization and vibration sense returned at that time.

Comment. This was a case of upward nystagmus on forward gaze in an alcoholic individual. The vertical nystagmus was accompanied by vertical oscillopsia. Since the patient became aware of oscillopsia and since nystagmus and oscillopsia disappeared simultaneously, it may be safely assumed that these symptoms were acquired and not of the congenital type. The patient showed another rare disorder in eye movements: spontaneous zig-zag or dissociated eye movements, which are sometimes seen in subacute encephalitis and often associated with marked, almost myoclonic, tremors throughout the body. It is remarkable that amytal and even alcohol, when given intravenously, abolished the upward nystagmus on forward gaze. The spontaneous, dissociated, almost choreiform, eye movements were also abolished by amytal.

Discussion

There are three points worthy of comment: (1) Nystagmus, (2) oscillopsia, and (3) effect of drugs on nystagmus and oscillopsia.

1. Nystagmus. Distinction must be made between vertical nystagmus on upward gaze and vertical nystagmus on direct forward gaze. Spontaneous vertical nystagmus on direct forward gaze is seen almost solely in cases of congenital nystagmus. The spontaneous vertical nystagmus on forward gaze found in our two cases, however, is of the acquired type. In each, the oscillopsia appeared shortly before admission to the hospital and both the oscillopsia and nystagmus on direct forward gaze subsided concomitantly with treatment.

Both of the patients were chronic, severe alcoholics who exhibited signs of encephalopathy and peripheral neuropathy. It is known that such patients have diffuse lesions throughout the entire nervous system and that the brain stem is one region which is frequently involved in cases of severe alcoholism. One of the symptoms of disease of the brain stem is vertical nystagmus on upward gaze. Clinicopathologically, cases of vertical nystagmus on upward gaze have been reported in patients with verified lesions of the colliculi, of the caudal part of the pons and the medulla at the level of the inferior olivary bodies, and, in fact, with lesions of the brain stem at almost any level.[1]

There have been few, if any, reports of clinicopathologic studies of cases of vertical nystagmus on direct forward gaze. To date, therefore, it is not possible to localize the lesion in these cases of vertical nystagmus on direct forward gaze with any degree of accuracy. One can only suspect that the pathologic condition is somewhere in the brain stem.

On the other hand, judging from animal experiments, it would appear that the lesion might be in the vermis of the cerebellum. Lesions in the posterior part of the cerebellar vermis in the experimental animal have resulted in spontaneous vertical nystagmus.[5]

Using cats, Spiegel and Scala electrocauterized the vermis to obtain "positional nystagmus." They also recorded that the animal exhibited vertical nystagmus on being observed in its "normal position" which is analogous to forward gaze in the erect human subject.[5]

This vertical nystagmus in the normal

position of the operated cats consisted of very slow oscillations—five jerks in 30 seconds in one instance. The fast component was generally upward, but in one experiment in which both globose nuclei, the pyramis and uvula, were destroyed, it was downward.

2. Oscillopsia. Oscillopsia is a symptom caused by movements of the eyeballs. It is usually apparent in the vertical plane. Vertical oscillopsia has been reported in cases of multiple sclerosis,[6] encephalitis, brain tumors, and vascular disease of the brain and brain stem.

Oscillopsia can be produced by vestibular stimulation. Vertical oscillopsia is frequently found when vertical nystagmus is induced by the Bárány chair or caloric tests. However, when horizontal nystagmus is induced, it is interesting to note that individuals tend to report their sensations as "dizziness" and omit the phenomenon of horizontal oscillopsia.

Horizontal oscillopsia is often seen in cases of congenital latent nystagmus. It becomes apparent when there is interference with binocular vision. Thus, when one eye is covered or a piece of cardboard is placed on the bridge of the nose so as to interfere with binocular vision, a marked horizontal nystagmus becomes manifest. With this horizontal nystagmus, the patient complains that the observed object shimmers or moves back and forth in the horizontal plane.

3. Effect of drugs. Despite the presence of gross nystagmoid movements of the eyes, oscillopsia is not found in cases of congenital horizontal nystagmus on forward gaze. There is, instead, a marked impairment of visual acuity. Under the influence of intravenous sodium amytal, the ever present congenital nystagmus can be arrested. This is followed by an improvement in the visual acuity.[3]

As the effect of the amytal wears off and the congenital nystagmus reappears, the patient complains of foggy vision. Interestingly enough, there is no oscillopsia at this latter time but instead the visual images may appear elongated in the plane of the nystagmus, or may appear multiple.[7]

In our cases, intravenous sodium amytal or alcohol by mouth temporarily abolished both the acquired vertical nystagmus on direct forward gaze, and the concomitant vertical oscillopsia.

Summary

Two cases of vertical nystagmus on direct forward gaze are described. Both of the patients complained of images bobbing up and down—vertical oscillopsia. When the nystagmus ultimately subsided the oscillopsia disappeared. Intravenous sodium amytal or alcohol temporarily abolished the vertical nystagmus and the concomitant vertical oscillopsia. Differentiation has been made between the acquired and the congenital types of vertical nystagmus on direct forward gaze.

477 First Avenue (16).

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TEMPORAL SUBCONJUNCTIVAL HEMORRHAGES AS A COMPLICATION OF RHINOPLASTIC PROCEDURES

WAYNE W. WONG, M.D.
Honolulu, Hawaii
AND
W. B. SLAUGHTER, M.D.
Chicago, Illinois

The occurrence of subconjunctival hemorrhages in one or both eyes as a minor complication of a rhinoplastic surgical operation is so consistent that this study was undertaken in an attempt possibly to explain some of the factors which cause these hemorrhages to be located in the temporal sector of the globe. It was thought that this study of 55 consecutive cases of rhinoplastic patients might also help to explain the frequent temporal location of the common spontaneous innocuous subconjunctival hemorrhages.

Only those cases that required a complete operation were selected. All operations were performed under local infiltrative anesthesia. The operating conditions and technique were essentially the same in the entire series.

In this series of 55 cases, subconjunctival hemorrhages were observed in 32 cases in one or both eyes. In 31 of these, the subconjunctival hemorrhages occurred only in the temporal sector of the bulb. Only in one patient were the hemorrhages other than temporal, this solitary case being circumlimbal in one of the eyes only. Even in this case, however, the hemorrhages were more intense in the temporal sector.

Table 1 shows the age, sex, degree of ecchymosis, and presence or absence of subconjunctival hemorrhages in these patients. The blood pressure was normal in all cases. No general systemic disease was found in the entire group. No local ocular inflammation was observed before, during, or after surgery. These cases were entirely elective and were done for corrective purposes.

From Table 1, it can be readily seen that the occurrence of subconjunctival hemorrhages is directly dependent on the degree of ecchymosis that is evident following surgery. The degree of ecchymosis as listed in Table 1 was divided roughly into three groups. It was considered minimal when there was a slight puffiness of the lower lid with only a faint discoloration which disappeared in a few days; moderate, when both lids were swollen and discolored to a greater degree; and pronounced, when both lids were swollen shut and the discoloration of the lids was violescent (fig. 1).

When subconjunctival hemorrhages complicate a rhinoplastic procedure, these factors are noted. Immediately postoperatively, the bulbar conjunctiva is not injected nor congested. There is no chemosis. The edema of the lids, in those cases that do have some reaction, is noted within 6 to 8 hours postoperatively. This is accompanied by a slight glassiness of the conjunctiva, with or without a slight congestion of the conjunctival vessels. This is due in part to the decreased sensitivity of the blinking reflex following the preoperative medication and the postoperative mental relaxation. No evidence of any subconjunctival hemorrhages is noted at this time.

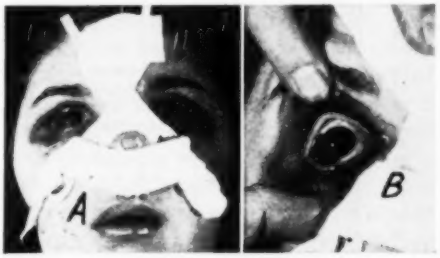

Fig. 1 (Wong and Slaughter). (A) Bilateral ecchymosis and bilateral temporal subconjunctival hemorrhages are present on the third postoperative day. (B) Close-up view of a typical subconjunctival hemorrhage.

TABLE 1

OCCURRENCE OF SUBCONJUNCTIVAL HEMORRHAGES IN RHINOPLASTIC PATIENTS

No.	Sex	Age	Ecchymosis	Subconjunctival Hemorrhages	Remarks
1	F	25	L.E. 3+	L.E.	Duration 3 weeks
2	F	18	B.E. minimal	None	
3	F	21	B.E. 2+	None	
4	F	27	B.E. 3+	B.E.	Duration 25 days
5	M	35	B.E. minimal	None	
6	F	18	Minimal	None	
7	M	18	B.E. 3+	B.E.	Duration 3½ weeks
8	F	21	R.E. 2+, L.E. 4+	L.E.	Duration 26 days
9	M	22	B.E. 4+	B.E.	Duration 4 weeks
10	F	26	B.E. 2+	L.E.	Punctate temporal
11	F	22	None	None	
12	F	23	Minimal	None	
13	F	23	R.E. 2+, L.E. 3+	B.E.	L.E. worse; duration 4 weeks
14	F	23	B.E. 3+	L.E.	Duration 20 days
15	F	15	B.E. 3+	B.E.	Duration 3 weeks
16	M	25	B.E. 3+	B.E.	Duration 3½ weeks
17	F	20	B.E. 2+	B.E.	Duration 4 weeks
18	M	28	Minimal	None	
19	F	23	B.E. 2+	R.E.	3 small postop. hem., L.E.
20	F	32	B.E. 3+	None	
21	F	30	R.E. 4+, L.E. 2+	R.E.	Duration 3 weeks
22	F	38	R.E. 4+, L.E. 3+	B.E.	R.E. more; and in nasal
23	F	32	R.E. 3+, L.E. 4+	L.E.	Duration 15 days
24	F	24	R.E. 2+, L.E. 4+	L.E.	Duration 15 days
25	F	26	R.E. 3+, L.E. 2+	B.E.	L.E. minimal
26	F	28	Minimal	None	
27	F	37	Minimal	None	
28	F	18	R.E. 4+	B.E.	Marked
29	F	25	Minimal	None	
30	M	25	Minimal	None	
31	F	45	Minimal	None	
32	F	42	B.E. 3+	None	
33	F	35	Minimal	None	
34	M	16	B.E. 3+	B.E.	
35	M	38	L.E. 2+	None	
36	F	24	B.E. 4+	B.E.	
37	M	30	L.E. 2+	None	
38	M	22	B.E. 3+	B.E.	Marked
39	F	26	R.E. 2+	R.E.	
40	F	30	Minimal	None	
41	F	26	Minimal	None	
42	F	20	R.E. 3+	B.E.	
43	F	25	Minimal	L.E.	Fractured right side
44	F	25	B.E. 2+	B.E.	Postop. epistaxis
45	F	30	B.E. 4+	B.E.	R.E. greater than L.E.
46	F	25	Minimal	B.E.	Very small
47	M	26	B.E. 3+	B.E.	
48	M	30	Minimal	None	
49	M	22	B.E. 2+	B.E.	Very small
50	F	24	B.E. 2+	B.E.	Small
51	F	18	Minimal	None	
52	F	28	Minimal	B.E.	Small
53	M	21	B.E. 2+	B.E.	
54	M	30	Minimal	None	
55	F	38	Minimal	None	

About 12 to 24 hours postoperatively, ecchymosis develops in one or both eyelids. The bulbar conjunctiva is not changed remarkably during this period. Immediate and constant use of ice compresses postoperatively does not appear to influence the development nor the degree of ecchymosis in those cases that subsequently develop ecchymosis of the lids.

At the 24-hour postoperative period, sub-

conjunctival hemorrhages have not yet appeared in all the cases that will eventually develop them. It is more common for the subconjunctival hemorrhages to appear between the 24th to 36th postoperative hour than between the 36th to the 48th hour. The general appearance of these subconjunctival hemorrhages when they first appear easily separates them into two distinct varieties.

In one group of patients, they form an intensely red triangular-shaped patch in the exposed temporal sector of the bulbar conjunctiva—the area delineated by the upper and lower lids as it meets the temporal limbus at about the 1:30 to the 4:15-o'clock positions. This area may or may not extend to the limbus, since there may be a bridge of clear conjunctiva between the limbus and the patch of hemorrhage. The hemorrhage may show some variation in intensity of color, but this is due mainly to the degree of oxygenation by the lacrimal secretion.[1]

On the second day of its appearance, there is a variable tendency for the hemorrhage to extend into the inferior fornix so that by the 72-hour period, the triangular patch may reach the limbus at the 6-o'clock position and may extend down into the inferior fornix, stopping at the inferior attachment of the palprebral conjunctiva. This downward extension is due to the massaging effect of the upper lid, as well as to gravity.

In a second group of patients, a less extensive type of hemorrhage is found. These occur as an irregular, horizontal, linear type of low-intensity hemorrhage and lie definitely in the 3- to 9-o'clock meridian. Composed of a small amount of blood, they are faint in comparison to the first type and their dense portion is usually closer to the limbus. The lateral extensions usually fade gradually into the normal surrounding conjunctiva. In width these hemorrhages are usually about 3 to 5 mm. at their widest portions. Such hemorrhages do not appear to be affected by the massaging effect of the lids nor by gravity.

The difference between these two types of hemorrhages is one of degree, although the causative factor and the location of the pathologic vessels are also different. In the first type, there appears to be a leakage from the deeper subconjunctival vessels; while in the second, there appears to be a diapedesis of the superficial conjunctival vessels. Ruedemann[2] describes these two types as (1) hemorrhages of obstruction and (2) hemorrhages of diapedesis.

The duration varies according to the amount of blood found subconjunctivally and the rate of absorption. Thus, some of these hemorrhages may be absorbed in 7 days while others may last as long as 4 weeks. The macroscopic rate of disappearance is unpredictable. A dense patch of subconjunctival hemorrhage may seem to remain stationary for many days but, in an equal number of days, the size of the hemorrhage may be rapidly reduced.

The direction of absorption is not always centripetal. In many cases, a clear patch of normal conjunctiva separates temporal patches of hemorrhage from the limbus, and this separation is maintained until the hemorrhages finally disappear. In other cases, the larger patches of hemorrhage are broken down into several smaller ones before they are finally absorbed. In most cases, however, the hemorrhage recedes slowly until there is only a small dot remaining at the 3-o'clock limbus. Massage does not appear to influence the rate of disappearance.

Studies[1] in the same patients of the resorption of subconjunctivally injected blood showed about the same rate of disappearance as spontaneously produced subconjunctival hemorrhages. However, these studies cannot be compared with the hemorrhages observed in our series since there were no disturbances of local circulation by adjacent surgical trauma.

Under the slitlamp one can readily see that the bulbar conjunctiva contains two layers of blood vessels.[2] The deeper layers of larger vessels are part of the branches of the anterior ciliary arteries and form a

part of the episcleral and scleral tissues. They anastomose rather freely with the superficial conjunctival vessels and penetrate the sclera about 3 to 4 mm. from the limbus. The superficial vessels are small and are located just beneath the conjunctival mucosa. There are approximately 2 to 3 times as many veins as arteries.

The difference in the size of the blood vessels between the deeper and superficial layers of conjunctival vessels forms the basis of the two types of hemorrhages. The large dense hemorrhage is from the deep vessels which are actually small arteries; whereas, the smaller, sketchy type of hemorrhage is from the superficial vessels which are arterioles and venules and are comparatively small. These hemorrhages are definitely venous in character. The greater number of veins in proportion to arteries in this region would leave the veins, according to the law of chance, more vulnerable to the forces of stress and strain.

If the superficial conjunctiva is touched with a moist cotton applicator or by the moving lids, it will move very freely over the deeper episcleral tissues. This freedom of movement appears to give the subconjunctival tissues an almost fluid appearance. It is in this rather loose areolar tissue that the subconjunctival hemorrhage occurs.

The dense type of hemorrhage, being from the deeper vessels, pours the blood from the bottom of this loose layer, therefore, making it impossible to locate the involved vessels. The thin sketchy type of hemorrhage empties the blood into this layer from the top of the areolar tissue. Here one can see small, faint patches of hemorrhage which appear as pinkish tufts located at the visible termination of the vascular tree. The confluence of these small areas of diapedesis produces a visible patch of subconjunctival hemorrhage.

The blood from the conjunctival vessels can be readily seen flowing toward the limbus in the normal conjunctiva. As these vessels are followed through an area of hemorrhage, their course stands out rather clearly because there are clear areas paralleling them. The older the hemorrhage, the easier it is to see these perivascular channels, which are areas of absorption and digestion of the subconjunctival hemorrhage.

Discussion

Circulation of conjunctiva

The circulation of the conjunctiva forms a part of the general circulation and is dependent on the general arterial pressure and on the general venous return flow. The arteries, capillaries, and veins, which form the circulatory units, are under the control of the vasomotor system that regulates the degree of constriction and dilatation of the vessels.

The local circulatory units can be subjected, therefore, to several kinds of disturbances.[3] (1) Vasomotor, which results from the change in neural control; (2) mechanical, such as those produced by mechanical obstruction of the lumen of the vessel either by a thrombus or an embolus within the lumen, by changes in the wall of the vessel, or by compression from without; and (3) biochemical, such as the action of split proteins from surgical trauma acting on the capillary wall or the action of surgical trauma depleting certain constituents from the body whose lack would in turn weaken the capillary wall.

The vasomotor disturbances have been shown by Ricker[4] to act in various degrees, depending on the strength of the stimuli. A weak stimuli affects the dilator fibers only and causes a dilatation of the vessels with an increase in the blood flow. A slightly stronger stimuli affects the constrictor fibers and causes a constriction of the arteries with a slowing of the circulation in the capillaries and veins. A stronger stimuli causes paralysis of the constrictor fibers and stimulation of the dilator fibers. At first this produces a dilatation of the vessels with an increase in the blood flow, but the dilator fibers soon become paralyzed and, above the dilated vessels, the artery is constricted. This results in

a slowing of the blood stream to complete cessation or stasis.

Depending on the action of the agents on the vasomotor system, three states of the local tissues can be distinguished. These are (1) active hyperemia with an increase in the blood flow, (2) passive hyperemia with a slowing of the blood flow, and (3) stasis or cessation of the blood flow.

Active hyperemia is produced by stimulation of the dilator fibers, the constrictor fibers remaining unaffected. Arteries, capillaries, and veins are dilated, and the blood flow is increased. There is, also, an enormous increase in the number of open capillaries with only a slight loss of fluid through the capillary wall.

Passive hyperemia with the slowing of blood flow occurs with the stimulation of the dilator fibers and a change in the constrictor fibers, which may either show a diminished activity or become completely inactive. In the region of the inactive constrictor fibers, the arteries, capillaries, and veins are dilated, and the circulation is slow and irregular. This form of hyperemia is characterized by an increased passage of fluid through the capillary walls and its accumulation in the surrounding tissues.

Stasis. When the central artery of the affected area becomes more constricted, there is still a greater dilatation of the vessels in the affected area and a still greater slowing of the blood stream results. Circulation is present but slowed. This form of vasomotor activity is characterized by the passage of red blood corpuscles through the capillary walls. They may pass through the capillary wall singly at one point, or hemorrhages may occur at a number of points. If the artery central to this area is markedly narrowed or completely occluded, the activity of the vasomotor fibers in the area is completely lost and, as a result, the vessels are relaxed and circulation is slowed to such a degree that stasis ensues. This latter state does not occur as a result of surgical trauma following rhinoplastic operations.

Obstruction to blood flow. The circulation of the conjunctiva can be disturbed mechanically by an obstruction to the flow of blood in the arteries or veins supplying it. When the obstruction is not complete and varying degrees of reflex constriction below the point of obstruction occur, there is a corresponding dilatation of the arterioles, capillaries, and venules distal to the point of constriction. The resulting condition produces a transudation of fluid and hemorrhages to a varying degree.

Mechanical obstruction of a vein is followed by a cessation of circulation in the vein and accompanied by a corresponding reflex constriction of the artery supplying the region. This occurs only in complete obstruction. Before circulation stops, however, there is first a slowing of the blood flow which produces a state of prestasis with its attendant hemorrhages. The immediate results of local circulatory disturbances are, therefore, a transudation of plasma into the tissues and attendant hemorrhages.

According to Landis,[5] the capillary blood pressure is capable of rising conspicuously during hyperemia (from 14 cm. of water to 23 cm. of water) and returns toward the lower resting level as hyperemia recedes. By the use of the micropipette introduced into the venous limb of a capillary at the base of the human fingernail, it was noted that, on cooling the skin at the base of the nail, a vasoconstriction resulted with a distinct drop in the capillary blood pressure. This, however, is followed in a few minutes by a secondary rise associated with a reactive hyperemia which has been studied so thoroughly by Lewis and his co-workers.[6] This mechanism may explain the nonbeneficial results of the ecchymosis reducing factor of ice compresses applied immediately postoperatively in our cases.

Another contributing factor in the production of these subconjunctival hemorrhages is suggested by an observation made by Hueck[7] that the protoplasm of the endothelial cells of the capillaries and the base-

ment membrane is capable of undergoing a transitory liquefaction to such a degree that it causes numerous openings to form and thus allows plasma and, in the case of larger openings, red blood corpuscles to pass through. This condition is produced in the state of prestasis, when the slowing of the circulation in the capillaries causes a diminution in the supply of oxygen and of nutrition to the walls of the capillaries. This same observation was also noted by Landis[5] in the capillary of the frog.

Physiology of capillaries

Most of the studies on the physiology of the capillaries have been made on the vessels themselves with little regard of the action of the contents of the vessels. The observations of Knisely and his co-workers[8] on the action of traumatized capillaries on the blood corpuscles and their subsequent effect on the capillaries themselves throw new light on the basic pathologic physiology of the blood vessels.

Briefly stated, Knisely discovered that, in injured tissue and blood vessels and in certain diseased states, the individual blood corpuscles become agglutinated and form clumps of cells. This mass of cells or "sludge" resists its own passage through the small vessels and consequently the rate of flow in these vessels becomes progressively slower. The "sludge initiating factor" at the site of injury continues to form more "sludge blood" which reduces the rate of flow through the capillary bed and slowly leads to various degrees of stagnant anoxia all over the body.

When this happens the walls of the postcapillary venules and small venules lose their ability to retain blood colloids. Anoxia of the endothelium is alone sufficient to cause this and severe anoxia always causes endothelium to leak rapidly.

One of the key facts in Knisely's observation is that blood coming through the arterioles of any organ may be studied as a valid sample of all flowing arterial blood in the body. He has used the uninjured bulbar conjunctiva of human beings in the study of "sludge blood" in various diseases.

It appears that one of the important factors in the production of subconjunctival hemorrhages is a local weakening of the capillary wall to any increase in intravascular pressure. The strength of the capillary wall is actually a measure of the intracellular adhesive power of the endothelial cells which make up the capillary wall.

This is, in turn, affected by such substances as rutin, vitamin P, citrin, and so forth, which are flavone glucosides in various degrees of purification, and factors causing endothelial anoxia. There is some question as to the mode of action of these substances and as to whether the tourniquet test which measures capillary fragility has any bearing on capillary hemorrhages.[9]

Wolffe and Danish[10] reported two cases in which subconjunctival hemorrhages occurred during the administration of rutin. In one case, the hemorrhage occurred about four weeks after continuous rutin therapy. In the second case, the hemorrhage occurred 24 hours after the starting of rutin therapy. In both of these cases capillary fragility tests, as measured by the Gothlin method, were normal.

The possibility that other causes may be concerned in the integrity of the normal cells must also be considered.[11, 12] It is highly improbable that the explanation can be found in a single agent or a group of similar agents but rather in a complex biologic reaction of which the flavone glucosides form only a part.

Structure of temporal conjunctiva

The almost constant location of these subconjunctival hemorrhages in the temporal sector of the bulb can be explained on an anatomic and functional basis. It is a well-known surgical fact that the structure of the temporal conjunctiva is much thinner than that of the nasal sector. The ocular movement of convergence, being a positive

function, places a stretching force on the conjunctiva of the temporal sector while that of the nasal sector is compressed into folds.

In addition, the anterior ciliary arteries that travel around the eyeball on the recti muscles are usually paired on all the recti muscles, except for the lateral rectus which usually carries one and occasionally no visible artery.[13, 14] The stretching force of ocular convergence on the temporal conjunctiva plus the strain of any venous obstruction would weaken this sector more than the others.

Biochemic effects of trauma

The biochemic effects of trauma have been studied in severe injuries, such as burns and fractures.[15] It seems probable that the formation of "sludge blood" is directly or indirectly concerned with the biochemic effects of trauma. Andreae and Browne[15] observed a rapid and marked destruction or utilization of ascorbic acid during the period immediately following injury.

Levenson and others[16] state that the stresses associated with severe injury are followed by a markedly increased turnover of protein and carbohydrate metabolism. Since it is known that the vitamins of the B group are intimately concerned with the metabolism of these substances, it is likely that the demand for and the utilization of them is likewise increased.

However, the trauma associated with the surgical procedure of rhinoplasty cannot be compared with cases of severe injury whose measurable demands on the body physiology are definite. Nevertheless, one may expect some of the physiologic forces to come into play, even though to a much lesser degree, in less severe injuries.

With all these factors contributing to the development of the temporally located subconjunctival hemorrhages in postrhinoplastic cases, it appears that the variable factor is the patient's reaction quotient to trauma. This particular aspect of the problem has been studied in the lower animals.[17, 18] The degree of postoperative edema in our cases would be a rough measure of the individual patient's reaction to trauma. When we consider this variable individual factor, the formation of these subconjunctival hemorrhages is the end result of a multitude of physiologic conditions.

Synopsis

The chain of events may be hypothesized in the following manner. The operative trauma causes an increase in the protein concentration of the lymph.[19] The formation of "sludge blood" now becomes a factor. This increase in protein concentration may not, in itself, change the permeability of the capillary walls[20] but, with a given capillary pressure, the colloidal osmotic pressure may vary to such an extent so as to influence capillary permeability.[5] Whether the permeability of the capillaries is changed reflexly or by the slowing of the blood stream by sludging, the total tissue anoxia and subsequent edema are increased. This postoperative edema causes a congestion of the angular veins and of the internal pterygoid plexuses so that the drainage from the external eye is affected directly and, possibly, reflexly.

The operative trauma may further deplete or utilize more rapidly certain substances which may have to do with capillary fragility. The subconjunctival hemorrhage is, therefore, a result of a disturbance in venous circulation when the postoperative congestion exceeds the intracellular adhesive power of the capillary wall. This congestion requires about 24 to 48 hours to create enough obstruction so that any sudden further increase in venous pressure, such as might be caused by coughing, laughing, or straining, plus the factor of ocular convergence, would be enough to put a strain on the weakest spot of the conjunctival vascular tree.

The single lateral rectus ciliary artery would have to carry a pressure load which in the other recti muscles is divided between two vessels. Moreover, the rare occurrence

of a spontaneous subconjunctival hemorrhage over the superior or inferior recti muscles is partly due to the compression and support afforded these areas by the upper and lower lids. The nasal and temporal sectors are subjected to an increase in the local vascular pressure. The structural weakness of the temporal conjunctival vascular tree is thus made evident by the typical temporally located subconjunctival hemorrhage.

Trauma per se is definitely not related to these hemorrhages. In a case in which a 12-mm. dermoid cyst was excised from the left supraorbital region under local infiltration anesthesia, a very large temporal patch of subconjunctival hemorrhage was noted within 24 hours postoperatively. There was a moderate amount of edema of the upper lid in this case, but the trauma incident to the removal of the cyst was certainly minimal.

In Case 43, in which it was necessary to fracture only one maxillary process, the subconjunctival hemorrhage occurred in the temporal sector of the opposite eye. The factor of a contrecoup force must be considered in this case, but it does not account for the absence of subconjunctival hemorrhages in those cases in which the apparent hardiness of the maxillary process required an excessive amount of trauma and yet no hemorrhages appeared. These observations seem to suggest that trauma in itself is not a sufficient cause but rather that the response of these patients to surgical trauma is the important factor.

Summary

1. In a series of 55 consecutive cases of rhinoplastic patients, 32 cases, or 58 percent, developed temporally located subconjunctival hemorrhages as a complication of the surgical procedure.

2. These subconjunctival hemorrhages were of two varieties, an intensely red triangular type and a streaky horizontally linear type.

3. Both of these types of hemorrhage appeared to be definitely related to the degree of postoperative ecchymosis and not to the degree of surgical trauma.

4. These hemorrhages always appeared from 24 to 36 hours following surgical trauma.

5. They were definitely venous in nature.

6. A discussion of the possible mode of formation and an explanation of their temporal location has been given.

1221 Victoria Street (34).

55 East Washington Street (2).

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OPHTHALMIC MINIATURE

To Mrs. Jane Mecom

London, July 17, 1771

Dear Sister,

. . . I thought you had mentioned in one of your letters a desire to have spectacles of some sort sent you; but I cannot now find such a letter. However I send you a pair of every size of glasses from 1 to 13. To suit yourself, take out a pair at a time, and hold one of the glasses first against one eye, and then against the other, looking on some small print.—If the first suits neither eye, put them up again before you open a second. Thus you will keep them from mixing. By trying and comparing at your leisure, you may find those that are best for you, which you cannot well do in a shop, where for want of time and care, people often take such as strain their eyes and hurt them. I advise your trying each of your eyes separately, because few peoples eyes are fellows, and almost every body in reading or working uses one eye principally, the other being dimmer or perhaps fitter for distant objects; and thence it happens that the spectacles whose glasses are fellows suit sometimes that eye which before was not used tho' they do not suit the other.—When you have suited your self, keep the higher numbers for future use as your eyes may grow older; and oblige your friends with the others. . . .

Your affectionate brother,
B. FRANKLIN.

NOTES, CASES, INSTRUMENTS

CILIA IN THE ANTERIOR CHAMBER*

With a report of a case

Olga Sitchevska, M.D., and
Brittain F. Payne, M.D.
New York

Cilia may enter the anterior chamber in perforating injuries of the eyeball, when they are cut or torn off from the lid margin and are carried into the anterior chamber either through the corneal or limbal wound. Occasionally the cilia may be implanted into the anterior chamber during surgical intervention for a cataract extraction.

The cilia sometimes are found lying free in the anterior chamber, but more frequently one end of the cilium is embedded in the iris. If the lens is injured one end of the cilium is stuck in the lens or lens capsule. In some cases the cilium is adherent to the corneal scar. Cilia are observed less frequently in the posterior chamber.

If cilia remain in the anterior chamber for a length of time, they may undergo certain changes, like splitting of the hair or separation of the cuticle. Blanching or depigmentation of the cilia may occur under the influence of the alkaline reaction of the aqueous. Wagenmann[1] stated that cilia may be occasionally absorbed by giant-cell formation.

Considering the frequency of perforating eye injuries, it is remarkable that cilia are not found more often in the anterior chamber. Sherman[2] gives an interesting explanation of this phenomenon. He believes that the reflex closure of the lid is usually delayed until the injury of the eyeball has occurred, so that the traumatizing object misses the contact with the lid margin. On rare occasions the closure of the lids occurs simultaneously with the impact of the injuring object which cuts the cilia and carries them into the anterior chamber. The injuring object in such cases frequently does not enter the eyeball as its force of motion is diminished by its friction against the lid margin.

Frequency of infection

Since cilia usually enter the eye in industrial accidents, in workers whose faces and eyelashes are soiled during the work, one could expect that the eyelashes would be a source of infection. This, however, does not occur frequently. It would seem that the rapid filtration of the aqueous from the anterior chamber allows little opportunity for bacterial growth there.

Lauber[3] believes that when an infection occurs in perforating injuries, it may have been brought about by the perforating object rather than by the cilia themselves. Müller[4] in culturing cilia of a number of working persons found no pathogenic microörganisms.

On the other hand, in a case described by Wagenmann[1] a cilium implanted in the anterior chamber caused a purulent iritis with a hypopyon 2.5 mm. high. After the removal of the cilium, the inflammation subsided promptly. Sympathetic inflammation was observed by Gunier[5] six months and by von Graefe[6] two weeks after the perforating injury with cilia implantation had occurred. Extraction of the cilia resulted in healing of the eyes.

Complications from retained cilia

While cilia may remain in the anterior chamber for a long time without giving rise to disturbing symptoms, they may sooner or later cause complications, such as epithelial tumors. The epithelium of the root sheath may become implanted, forming a cyst.

* From the New York Eye and Ear Infirmary.

"The implantation cysts thus formed may be of two distinctive types: solid looking, round or oval tumors, so-called pearl cysts, or translucent cysts with thin walls" (Duke Elder[7]).

Implantation cysts of the iris associated with the intrusion of an eyelash into the interior of the eye were reported by Bonnet and Paufique,[8] and Roth and Geiger[9] in whose case the injury was caused by a lead pencil, by Krachmalnikov,[10] Moore,[11] and by Horay.[12]

Cilia in anterior chamber

Cilia in the anterior chamber are not common. Popov[13] states that only two such cases have been seen among 80,000 eye patients in the Rostov Clinic and only one case in three years was found among 43,471 patients in the Astrakhan Clinic. Müller[4] reported that only five cases of cilia were observed in Fuchs's clinic in Vienna among 30,000 new patients. Sharpe[14] in his review of the literature, in 1925, stated that 75 similar cases were reported within the last 100 years.

At the New York Eye and Ear Infirmary, out of a total number of 374,721 eye patients examined during 15 years (1932 to 1947), only two such cases (including ours) have been recorded. These data confirm the infrequency of penetration of cilia into the interior of the eye.

Reports in the literature indicate that cilia may remain in the anterior chamber for a number of years without causing inflammation. Gradle[15] examined a 24-year-old man for blepharoconjunctivitis. There was a history of an injury to the right eye with scissors at the age of five years. The slit-lamp examination showed the presence of a depigmented cilium in the anterior chamber. The cilium has been there for 19 years without evidence of inflammation.

Hughes,[16] while refracting a naval officer, discovered an eyelash in the anterior chamber. A history was obtained from the patient that he was injured in the eye with a fountain pen when he was a child.

Roll[17] reported a case of a cilium being adherent to the opaque lens for a period of 19 years. Koenigstein[18] and Guzman[19] observed a cilium in the anterior chamber for 20 years. Henneberg[20] reported that inflammatory symptoms appeared 15 years after a perforating injury of an eye with the implantation of a cilium. Sharpe[14] reported a case of a cilium in the anterior chamber for 33 years. Schwartz[21] and Müller[4] each described a case of eyelash in the anterior chamber for 34 years. The eyes remained quiet all that time.

Cases of more than one cilium

It is evident from literature that cases of cilia in the anterior chamber are rare, and cases in which more than one cilium is observed are still rarer. Shagov[22] discovered two cilia in the anterior chamber after a magnet extraction of a piece of steel from the eye. An attempt to remove the cilia was unsuccessful as they moved to the posterior chamber. They remained there for a two-year period of observation.

Lichtner[23] and Payne [24] removed two cilia from the anterior chamber after a perforating injury of the eye. Franklin and Cordes[25] found three cilia and Sharpe[14] and Begle[26] have observed four eyelashes in the anterior chamber.

Bulson[27] and Valude[28] each reported a case of five cilia entering the interior of the eyeball after a perforating injury with a piece of wire. Mikhailov[29] reported a case of six cilia in a young woman's eye which was hit with a piece of wood; they were embedded in the iris. Three cilia were removed with an iris forceps with ease through an incision at the limbus, while the other three were removed with great difficulty and a cataract formed a week later.

Removal of cilia

The removal of the cilia from the anterior chamber is by no means a simple procedure, particularly when more than one

cilium is implanted. A number of complications may occur: the collapse of the anterior chamber because of the rapid outflow of the aqueous, the tendency of the iris to prolapse, or a hemorrhage from the iris which obscures the operating field. The most serious danger is the injury to the lens. Paderstein[30] reported a case in which he removed a cilium 5 weeks after a penetrating wound of the eyeball; 8 weeks later another cilium was seen and the attempt to remove it was frustrated by a hemorrhage from the iris with the lens becoming cataractous a week following the operation.

Fig. 1 (Sitchevska and Payne). Photograph of slitlamp drawing of two cilia implanted in the anterior chamber following a perforating injury of the left eye. The cilia, embedded in the iris, are enveloped by an exudate.

Report of a case

History. E. J., a Negro girl, aged 19 years, was first seen on March 4, 1948, one-half hour after an injury to the left eye. While working on a toy, she was struck in the left eye by a fine piece of wire from another toy a nearby worker was handling. She experienced a sharp pain in the eye and the vision became blurred at once.

Eye examination. There was marked photophobia and ciliary injection of the left eyeball and a gaping wound of the cornea at the 12-o'clock position, 4 mm. below the limbus. The anterior chamber was filled with clotted blood, mixed with grayish shreads of the turbid aqueous; the fundus could not be seen. Vision was reduced to hand movements. The right eye was normal and the visual acuity was 20/15.

The patient was hospitalized at the New York Eye and Ear Infirmary. She was given cold compresses, drops of a solution of atropine sulphate, and intravenous injections of triple typhoid vaccine. Three days later the hyphemia was absorbed.

The slitlamp examination showed that the corneal wound was sealed and the anterior chamber was restored. The aqueous was still somewhat cloudy. A square corneal opacity was forming at the site of the injury and there was another deep-seated opacity in the cornea at the 6-o'clock position, 3 to 4 mm. above the limbus, which extended toward the temporal side of the cornea.

The striking feature of the slitlamp examination was the presence of two thin, long foreign bodies in the anterior chamber which were recognized by one of us (B. F. P.) as cilia. The cilia were embedded in the pupillary margin of the iris at the 7-o'clock position. One cilium was located at the anterior border of the iris; while the second seemed to be behind in the posterior chamber. They were surrounded with a newly formed grayish exudate.

The free ends of the cilia were extending obliquely across the dilated pupil at about axis 70°. A small opacity in the lens capsule near the embedded cilia was observed. A posterior synechia had formed, and the pupil was of irregular shape at this site (fig. 1).

The roentgenogram showed no intraocular foreign body. The eye quieted down; vision improved to 20/50; and the patient was discharged from the hospital three weeks after admission.

Course. Four weeks following the injury, the patient complained of pain in the eye. Photophobia and ciliary injection were present and a flare in the anterior chamber was observed on slitlamp examination. She was re-admitted to the hospital and was operated on April 6, 1948 (5 weeks after injury), under local anesthesia for the removal of the cilia.

Operation. A keratome incision was made at the 9-o'clock position at the limbus and the incision was enlarged with scissors on either side. A Hunt capsular forceps was used to enter the anterior chamber, and one cilium was removed with ease. It was difficult to remove the second cilium, however, and several attempts were made before it was finally grasped and removed. A small peripheral iridectomy was done in order to prevent prolapse of the iris.

At the conclusion of the operation a pigmented, filmlike emulsion filled the anterior chamber, which had to be irrigated. This probably was free pigment from the pigment layer of the iris. Postoperatively, the patient was put on intramuscular injections of penicillin. Recovery was uneventful.

At examination, five months after the injury, the eye was quiet, an opacity of the cornea was seen below the limbus at the site of the entrance wound and another one was present at the 9-o'clock position, the field of the operation. A third pigmented corneal opacity was deeply seated about 4 mm. above the limbus at the 5-o'clock position. The opacity of the lens capsule remained unchanged. The vision of the eye was 20/50, unimproved.

Conclusion

A case of two cilia in the anterior chamber following a perforating injury of the eye and their successful removal is described. The fine piece of wire must have hit the eye and lid with sufficient force to cut off the cilia and carry them into the anterior chamber, injuring the cornea below from the endothelial side and causing no injury to the lens itself, which is rather remarkable in a penetrating injury of this type.

The frequency of occurrence, the number of eyelashes in the interior of the eye, the complications encountered during the operation for their removal are discussed.

Although the cilia may be retained for a number of years in the anterior chamber without giving rise to symptoms, it is our belief that an attempt should always be made at the earliest possible moment to remove them because of various threatening complications, chiefly that of the formation of epithelial cysts.

30 Fifth Avenue (11).
17 East 72nd Street (21).

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POSTOPERATIVE WOUND INFECTION CURED BY PENICILLIN*

BEULAH CUSHMAN, M.D.
Chicago, Illinois

History. Mr. H. J. H., aged 38 years, came to the late Dr. G. F. Suker in September, 1931, with the history of attacks of blurred vision in the right eye, lasting for 10 minutes to one hour, for three years. Vision in the left eye had become blurred for the first time a few days earlier.

A diagnosis of chronic glaucoma was made, and deep root iridectomies were done on both eyes in October, 1931. Miotics were necessary in the right eye after a few weeks and in the left eye after a few months.

Tension in the right eye gradually increased and remained around 35 mm. Hg (Schiötz), with a total loss of vision in 1937. The tension in the left eye was irregular at intervals, but it always responded satisfactorily to the use of various miotics. Prostigmin solution and tablets by mouth were found to be the most satisfactory treatment when the patient became sensitive to pilocarpine after 1940.

* From the Department of Ophthalmology, Northwestern University Medical School.

Course. In 1944, the refraction gradually changed to a compound myopic astigmatism and there was a mild iridocyclitis of the right eye. An incipient cataract was present in the left eye. In April, 1946, the diagnosis of diabetes mellitus was made. This condition was controlled with diet.

Operation. On August 14, 1946, Dr. Der-

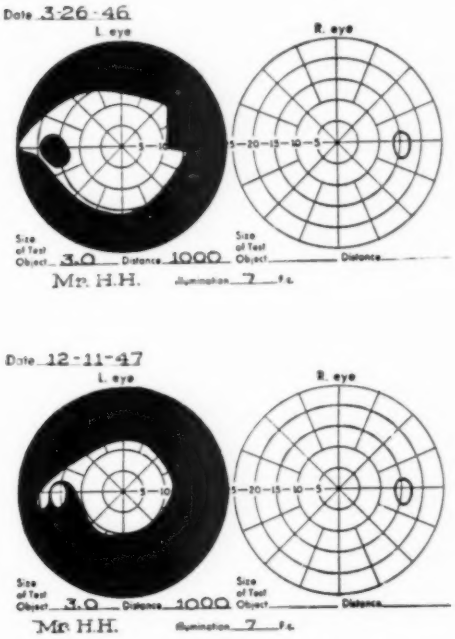

Fig. 1 (Cushman). Visual field studies before and after cyclodialysis and intracapsular lens extraction in the left eye.

rick Vail did a cyclodialysis of the left eye preliminary to an intracapsular lens extraction in this eye. The lens extraction was done the following week.

On the day following the operation, pain was complained of during the early morning, but no pain was present at the time of rounds, and the dressing was not changed. On August 16th, when the dressing was changed, an exudate was present along the corneal wound and in the anterior chamber. The conjunctiva was swollen and the upper lid edematous. Scattered, superficial abscesses were present in the skin of the upper lid.

Infection and treatment. A smear was positive for Staphylococcus hemolyticus, and later the culture was reported positive. Penicillin (1,000 units) was dropped into the eye immediately, and 10,000 units were given intravenously every three hours. Typhoid vaccine (15,000,000) was given.

The following day, penicillin was instilled into the anterior chamber after removal of 0.1 cc. of aqueous by means of a fine needle introduced through the recent incision. Sulfadiazine and soda bicarbonate (10 gr. every 4 hours) were given.

Improvement of the eye was immediate following the instillation of penicillin into the anterior chamber, and healing proceeded most satisfactorily.

Outcome. Vision in the left eye was: October 21, 1946—fingers at three feet; left pupil drawn up; April 14, 1947—iridotomy; June 24, 1947—20/16 with a +11.0D. sph. +4.0D. cyl. ax. 160°; with a +4.0D. addition, 4-point type.

The visual field of the left eye was contracted to 20 degrees with a 3/1,000 test object. There was complete cupping of the left disc. Tension in the left eye was 19 mm. Hg (Schiøtz).

25 East Washington Street (2).

SOME OCULAR EFFECTS OF THE SYSTEMIC ADMINISTRATION OF ANTIHISTAMINICS

JOSEPH V. M. ROSS, M.D.
Berwick, Pennsylvania

The use of antihistaminics is becoming an increasingly popular procedure in everyday medicine and a rather complete knowledge of the side-effects of such drugs seems to be desired. Ocular side-effects have been recorded often in the literature and in a review of 12 articles[1-12] I found them men-

tioned in 6. Some of the authors feel that most antihistaminics possess an atropinelike or vagal-paralyzant effect. There seems to be some disagreement as to whether or not oral administration might give pupillary dilatation and reduce the capacity for accommodation as does topical application. Blurred or dim vision is the ocular complaint mentioned. In one report in which a shocklike effect followed the oral administration of an antihistaminic, it was not clear if the origin of the blurred vision was cerebral or not.

During the past two years I have seen four cases in which the use of an antihistaminic resulted in ocular complaints, and which bring up some problems that warrant reporting.

Case reports

Case 1. A. G., a woman, aged 38 years, was receiving therapy for a limbal type of vernal conjunctivitis. Before medication was given, vision was normal for near and far with or without the correction (determined by cycloplegia): O.D., +0.5D. sph. ⌒ −0.5D. cyl. ax. 175°; O.S., +0.25D. sph. ⌒ −0.25D. cyl. ax. 170°. The patient received 100 mg. pyribenzamine daily for 13 days, at the end of which time she complained of blurred vision for far and near with difficulty in reading. Vision was 20/30, O.U., at far and 8-point type was read at 14 inches, O.U. Refraction revealed the following: O.D., +0.25D. sph. ⌒ −1.0D. cyl. ax. 175° = 20/20; O.S., −1.5D. cyl. ax. 170° = 20/20. An addition of +1.5D. sph., O.U., was necessary to give normal near vision.

With a contact lens in place, the following was found: O.D., +0.25D. sph. ⌒ −0.5D. cyl. ax. 175°; O.S., −1.0D. cyl. ax. 170°.

The amplitude and range of accommodation were diminished and the refractive changes noted, but all other ocular findings were normal. With cessation of the medication all symptoms disappeared and the findings at a later date were the same as at the early visits.

Case 2. J. M., a man, aged 65 years, had relative visual acuity of 20/50, O.U., and 0 at 14 inches, O.U., corrected to normal at near and far. The patient had chronic asthma and, after having taken 100 mg. of pyribenzamine daily for one month, complained of blurred vision for near and far.

Ophthalmic survey was normal except for vision of 20/40, O.U., at 6 meters with corresponding near vision, not correctible. There was a diffuse corneal edema similar to that seen following the ingestion of atabrine and an increased corneal relucency. The refraction (which was done with difficulty) was the same as found previously. All symptoms cleared when the drug was stopped; all findings were normal in one week.

Case 3. W. F., a man, aged 55 years, presented a bilateral contact dermatitis of the lids. Both near and far vision were easily corrected to normal. Eight days after taking 75 mg. of pyribenzamine daily he complained of blurred vision, especially for near. The patient saw 20/20 O.U., however, but only read 8-point type at 14 inches instead of 4-point, but with a +3.25D. addition, O.U., instead of the +2.25D. he was wearing, read normally at near.

All other findings were normal except for the finding of a few vitreous opacities which were not noted previously. Refraction for distance was the same as previously. In one week after cessation of the drug, the patient had no complaints and the findings were as they had been at the first visits. The vitreous opacities persisted, however, and the patient was now conscious of them.

Case 4. G. H., a boy, aged 11 years, presented a limbal type of vernal conjunctivitis for which pyribenzamine was given (100 mg.) for two days. Relative visual acuity was 20/25, O.U. Cycloplegic refraction then revealed the following: O.D., +0.5D. sph. ⌒ −2.75D. cyl. ax. 7° = 20/20; O.S., +0.5D. sph. ⌒ −2.5D. cyl. ax. 175° = 20/20. The postcycloplegic findings two weeks later were the same, but the patient

complained of a mist before the eyes. Now, only 20/25, O.U., vision could be attained. The pyribenzamine was discontinued and two weeks later the refraction with cycloplegia was: O.D., +0.25D. sph. ⊂ −0.5D. cyl. ax. 18° = 20/20; O.S., +0.5D. sph. ⊂ −0.5D. cyl. ax. 172° = 20/20. All other findings were normal throughout.

Summary

Four cases are presented in which there were ocular side-effects from the systemic administration of antihistaminics. The condition for which the drug was originally administered was not benefited in any case. The complaint in all these cases was blurred vision for far and near. The mechanisms involved were varied and all the reasons are not clear at this writing. In no case were the pupillary reflexes disturbed.

In Case 1, there was a depression of accommodation as well as refractive changes. Use of the contact lens suggested that these changes were due, at least in part, to factors other than corneal. The refractive changes were similar to those in Case 4.

In Case 2, that of a patient who had been asthmatic for years, there was seen a diffuse corneal edema with an increase in corneal relucency and no other significant changes.

In Case 3, a depression of accommodation only was noted. The question of the vitreous opacities is not settled.

In Case 4, an extreme refractive change occurred and the patient needed high minus cylinders at an axis of nearly 180°.

All changes abated with discontinuance of the drug, and the drug was not readministered to test specificity of action. The exact incidence of ocular side-effects was not determined.

Discussion

The ophthalmologist should certainly include in his history a question as to whether the patient is taking antihistaminics. In some cases, these drugs can cause blurred vision for near and far. The factors as determined in these cases were corneal edema, refractive changes, and depression of accommodation. Apparently factors other than corneal are involved in the refractive changes although corneal changes might likewise occur. The depression of accommodation might well be explained by the atropinelike action of the drug. The presence of vitreous opacities in one case brings up the possibility of vitreous changes.

321 East Front Street.

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A NEW ORBITAL IMPLANT

ORWYN H. ELLIS, M.D.
Los Angeles, California

Many new orbital implants for use following enucleation have been developed during and since World War II and much of the credit for stimulating interest in this work must go to Cutler.[1, 2] A new type of completely covered implant, which embodies a combination of sound surgical and prosthetic principles, is presented in this report. The indications for enucleation and evisceration will not be discussed, and only minor points in the enucleation procedure, as they affect the orbital implant, will be presented.

SURGICAL ANATOMY

As is well known, Tenon's capsule and the conjunctiva are fused with the sclera for a distance of 1 to 2 mm. about the limbus. The normal cornea has an average diameter of between 11 and 12 mm. Thus in doing an enucleation, approximately 16 mm. of circumference are lost.

The exact size of a sphere which can be inserted into the reduced space of Tenon following an enucleation can therefore be computed by subtracting the lost circumference from the circumference of the eyeball. In an adult a round implant of more than 18 mm. in diameter will stretch Tenon's capsule, and I agree with Fralich[3] that those surgeons recommending an implant of 20 to 21 mm. in diameter are forgetting the size of Tenon's space after enucleation.

When Cutler basket implants were used, I found that they were frequently extruded and that, when retained, considerable atrophy developed above and behind the implants. It was also noted, especially in children, that the conjunctiva pulled into the cavity was at the expense of the conjunctiva of the cul-de-sacs, especially the superior. A prosthesis that could be inserted had, therefore, to be narrow with very little motion. It is a well-established fact that the prosthesis will move in direct proportion to the movement of the cul-de-sacs, provided transfer of motion from the implant stump to the prosthesis is successful. If atrophy or reduction in size of the cul-de-sacs takes place, motion of the prosthesis is correspondingly reduced.

NEW IMPLANT

When Cutler's basket implants were placed in orbits, I noted that the prosthesis made after the socket-mold technique moved about the same whether or not the peg or stud was added to the prosthesis. A plastic basket was then made increasing the depth with plastic, with the idea that the muscles could be attached to the rim or tied through the holes and that the rim and sides of the implant would transfer motion to the prosthesis.

Later it was found that, by increasing the amount of plastic posteriorly so that the implant was essentially a plastic sphere with a rim, the prosthesis could be fitted to the sides where the covered implant transfers motion most readily to the prosthesis (fig. 1) and that, by so doing, motion could be increased.

PROCEDURE

The procedure of choice as it evolved is: The conjunctiva is incised as close to the limbus as possible. The conjunctiva and Tenon's capsule are then freed from the globe by blunt dissection. One rectus muscle is isolated and Tenon's capsule is stripped well back. A double-armed chromic catgut

suture is placed in the tendon, adding a cinch suture in the lateral band of the tendon, similar to the muscle stitch of Wiener.[4] The tendon is then cut from the globe. In succession the tendons of the other three recti muscles are similarly sutured and cut. The enucleation is completed and the hemorrhage controlled.

Next, the ends of each suture are passed through the corresponding holes under the rim of the implant and the tendons are tied together, medial to lateral and superior to inferior. If desired, the tendons may be attached to the rim in the manner described by Cutler.[2] However, to date and in my hands, Cutler's technique has seemed to prolong the procedure unnecessarily.

A running purse-string suture of white silk is then placed in Tenon's capsule and pulled tight, completely covering the implant. It was found that Tenon's capsule must be separated well back from each muscle before cutting it loose from the globe, otherwise the operator will be unable to close the capsule completely over the implant. A simple running black silk, or interrupted sutures, are used to close the conjunctiva in a horizontal line.

When the optic nerve is cut, a perforation of the capsule is made posteriorly through which an implant could be forced if one too large were inserted. Irvine[5] recommends suturing this posterior hole. He finds that this gives good support, since it brings the implant well forward. However, this method results in a considerable reduction in the size of the remaining Tenon's space, and it is therefore necessary to use an implant of smaller size.

Postoperatively, with the new implant, there has been surprisingly little edema, no more than with the routine enucleation using a glass-ball implant without muscle sutures. The original pressure dressing should be left in place 5 to 7 days, if possible. By this means postoperative edema is kept to a minimum. In no case has the conjunctiva bulged out between the lids. These patients are usually ready for a temporary prosthesis before two weeks.

Discussion

When a glass-ball implant was used, the implant stump usually moved well in all directions, but the stump did not transfer its

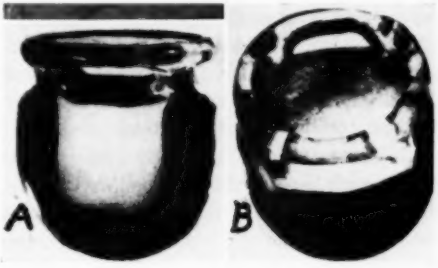

Fig. 1 (Ellis). A new orbital implant. (A) Side view. (B) Top view.

motion to the prosthesis. With this new implant, the movement of the ring is transferred directly to the prosthesis, and the lag of motion frequently seen in covered implants has not been noted.

The implant is made in varying sizes. In most adult orbits the large (18 mm.) size fills Tenon's space nicely but does not distend it. The implant is of the proper depth so that the covered rim protrudes 2 to 4 mm. which is adequate to provide flat sides for the proper fitting of the prosthesis. The inner surface of the prosthesis is molded to fit as snugly as possible around the outside of the projecting rim and sides of the implant. In this manner it is possible to avoid pressure on the conjunctiva, the weight is distributed evenly and, since the prosthesis does not rest on the lower lid, sagging is eliminated. After the prosthesis is fitted a wide excursion is present, but movements in the extremes of rotation are not necessary.

The status of implants that are not completely covered with conjunctiva has not been fully settled. A number, even under the most favorable conditions, are not re-

tained. Frequently, the muscles pull loose from the implant. When the implant herein reported is used, the muscles are attached with ease, and they aid in maintaining the implant in its proper position in the orbit.

Sensitivity to acrylic has occurred following use of implants not completely covered by conjunctiva. In some instances, apparently, a foreign-body reaction has taken place. No case of sensitivity to this new implant has come to my attention. The fact that it has been made only from Lucite may account for the lack of reaction.

To date a total of about 80 of these implants have been used by a number of ophthalmologists besides me, all of whom are enthusiastic about the ease of insertion, the normal appearance of the prosthesis, and the extensive excursion usually present when this implant has been used in conjunction with a prosthesis experdly made by the socket-mold technique. In only one case has there been extrusion or migration of the implant. A number have now been in place for two years. In no case has the implant changed position, orbital atrophy has not occurred as yet, and complications concerning the prosthesis have been few and of minor importance.

Since the introduction of this implant, quite a number of modifications have appeared. In some instances, the rim has been omitted; in others, the rim has been flattened. These changes, in my opinion, are steps backward. The Cutler basket implant taught us one thing—that the rim will transfer movement from the implant to the stump which, except in rare instances, the round implant will fail to do. The implant herein presented is certainly not perfect. It is in a stage of development from which, it is hoped, an implant that will satisfy all specifications may be evolved.

When the procedure as presented is used with the new implant, uniformly good results will be obtained. The method is not technically difficult and requires little additional time to that of routine enucleation with implant. One of the most important factors in orbital implants is permanency, and to date the completely covered implant herein described gives every promise of fulfilling this requirement.

523 West Sixth Street (14).

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Historical miniature

Egyptian Ophthalmology

The "rising of water in the eye" was treated with a preparation of lapis lazuli, verdigris, balsam, milk, and crocodile earth. Ebers interprets the name of the disease as cataract, but Hirschberg agrees with Lüring that intractable epiphora is meant.

Hirschberg, *Graefe-Saemisch Handbuch.*

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

WASHINGTON (D.C.) OPHTHALMOLOGICAL SOCIETY

January 3, 1949

DR. JEROME A. SANSOUCY, *president*

ABNORMALITIES OF THE OPTIC DISC

DR. FRANK B. WALSH, Baltimore, Maryland (by invitation) spoke on "Abnormalities of the optic disc; their importance in diagnosis," and illustrated his very interesting talk with numerous slides. Among others, the conditions that excited most interest were pseudoneuritis, aplasia of the optic nerve, hyaline bodies of the disc, holes in the disc, and the ophthalmoscopic differentiation of papilledema and optic neuritis.

Dr. Walsh described some of the abnormalities of the optic disc which cause confusion in diagnosis. Most of the anomalies selected for consideration have been confused from time to time with papilledema associated with increased intracranial pressure.

His criteria for the diagnosis of papilledema associated with increased intracranial pressure were: (1) Absence of pulsations of the vein on the disc, (2) measurable amount of papilledema, (3) the presence of exudates or hemorrhages, (4) presence of pallor of the swollen disc, (5) marked narrowing of retinal vessels, and (6) transient blurring of vision.

The ophthalmologist should be the examiner, Dr. Walsh stated, who briefly refutes the diagnosis of papilledema, and explains the presence of the apparent choking in conditions like persistent Bergmeister's papilla, hyaloid cyst, hyaline bodies, high hyperopia, and tilting of optic discs. He placed particular emphasis on the subject of hyaline bodies in the discs, their appearance, and the field defects with which they are sometimes associated. Costen was quoted as saying that there seems no question regarding progress of the field defects.

Dr. Walsh's paper was discussed by Dr. Benjamin Rones, and questions were proposed from the floor by Mrs. Helenor Wilder, Dr. Sterling Bockoven, Dr. David Cogan, Dr. Edward Cummings, Dr. O. H. Fulcher, Dr. Joseph Kemler, Dr. H. V. Rizzoli, Dr. Ernest Sheppard, Dr. G. Victor Simpson, Dr. Harold Stevens, and Dr. Jonathan M. Williams.

Thomas A. Egan,
Secretary-Treasurer.

COLORADO OPHTHALMOLOGICAL SOCIETY

September 18, 1948

DR. RALPH DANIELSON, *presiding*

PERSISTENT HYALOID ARTERY

DR. WILLIAM KUHLMAN presented M. V., a girl, aged 10 years. She had been an eighth month premature, weighing 3½ pounds at birth. At 3 months she had an obstructive jaundice. At present she is well, but has spastic movements and is mentally subnormal. Vision in the left eye is 20/100. The left lens is clear. Extending from the disc to the lens is an entire central hyaloid artery in a thin membrane. The artery breaks up into branches as it nears the lens.

KERATITIS WITH IRIDOCYCLITIS

DR. VON HALLER BROBECK and DR. SAM BROWN presented Mr. N. C. R., aged 57 years. On December 9, 1946, he was seen with a corneal ulcer with keratitis. The ulcer was cauterized with iodine and the eye kept atropinized. The ulcer healed, leaving two scars in the center of the cornea.

On May 11, 1948, he returned, again complaining of pain in the right eye. Vision in the right eye was 20/40. A central keratitis was present. No history of tuberculosis was elicited. Physical examination was negative. Reaction to tuberculin was markedly positive. The pupil was dilated with atropine and typhoid-H antigen was given intravenously. In June, since no improvement had taken place, a course of penicillin treatment was given, and whole blood was instilled into the eye. An iridocyclitis developed. Salicylates were given orally. The vision decreased to perception of large objects only. The thermophore was used on the cornea. In August, an abscess developed in the anterior chamber. A keratotomy was performed and culture of the pus showed Staphylococcus albus. Since then there has been no pain but the eye continues very red. Vision is reduced to seeing large objects.

Discussion. Dr. William Kuhlman said that he had had this patient in the hospital for 10 days under streptomycin therapy. The eye cleared while under treatment. Perhaps, if it had been continued, there would have been more clearing.

Colonel Martin said that, in his experience, while streptomycin is being given, the eye clears but, when it is stopped, the infection starts up again. He considers that the bacterial invasion has been stopped but no immunity built up. He uses 1 gm. a day in divided doses for 30 days, than 0.5 gm. a day for 90 days.

Dr. George Brown suggested X-ray treatment. Dr. James Rigg said that carbuncles clear when penicillin is injected in the tissue around. He suggested retrobulbar and circumcorneal injection of streptomycin or penicillin.

Dr. George Stine saw the patient in consultation two years ago. It was a compensation case, the cornea having been scratched by a box while the patient was at work. At that time he had a small, irritable, sharply delimited opacity. It is a bizarre keratitis, probably a tuberculous process.

Dr. Morris Kaplan had tried injecting penicillin into the anterior chamber but a severe reaction had taken place. Colonel Martin suggested that it might be possible to introduce penicillin into the anterior chamber by iontophoresis. This treatment has produced a reasonable clearing of a sclerosing keratitis of 11 years' duration. It is useful in corneal and iritic lesions.

Band-shaped keratitis

Dr. von Haller Brobeck and Dr. Sam Brown presented Mr. J. S., aged 36 years. The right eye has had an opacity since childhood. About eight years ago a similar opacity began to appear in the left eye. It had become much more dense in the past year. Vision is: R.E., 20/60; L.E., 20/50, with or without glasses. Each cornea has a dense horizontal band across the center with clear cornea above and below. Each iris is markedly atrophic. On June 25, 1948, the scar was resected. During the operation, the cornea was perforated and a conjunctival flap was drawn over it. When the flap retracted, a pseudopterygium remained. On August 9, 1948, this was removed. Following this, a good cosmetic result was obtained. On September 3rd, nutrient vessels leading into the scar were cauterized with trichloracetic acid. At this time a descemetocele was beginning to form in the upper outer area of the cornea.

Central choroiditis

Dr. F. Nelson presented Mr. R. H., a Negro aged 46 years. One year ago he was seen by several other oculists over a period of several months because vision in the right eye had started to blur. No objective pathologic condition was found at that time. When the patient was seen on June 17, 1948, vision of the right eye was reduced to 5/18, partly. A central scotoma was present. Ophthalmoscopically an extensive destructive process was found in the right macula which appeared to be punched out. A sausagelike hemorrhage surrounded the macular region below and nasally. This hemorrhage was

subretinal and the vessels could be seen climbing over it. An incomplete white star figure was between disc and macula and below the macula. The left eye was normal.

Physical examination revealed several dental abscesses, the largest one, located above the upper right molars, measured 2.5 by 1.5 cm. All pus cysts were removed from the patient's jaw and the retinal process then cleared up gradually. On August 20th, vision was 5/15, partly.

In cases of central scotoma with deterioration of vision and no evidence of retrobulbar neuritis the macular region of the affected eyes must be checked over a long period of time. A search for focal infections is especially indicated in such cases.

CENTRAL CHORIORETINITIS

DR. GEORGE H. STINE and DR. KATHARINE H. CHAPMAN presented two cases of old healed central chorioretinitis in which the patients were not conscious of having any trouble. K. K., a boy, aged 15 years, first noticed last summer that his right vision was blurred. He had never had any discomfort in his eyes. He has always shot with the gun at his left shoulder. He writes with his left hand. At about three years of age he had a severe pneumonia. Vision is: R.E., 20/200; L.E., 20/12. The right macula has an area of choroidal atrophy, 1 p. d. in diameter, with irregular pigmented margins.

Mrs. C. G., aged 31 years, a housewife, had noticed some dimness of vision with pain in the left eye for a few days about 10 years ago. No oculist was consulted at that time, and only recently had she noticed that she could not see to read with the left eye. Vision is: R.E., 20/25; L.E., 4/300. The left eye has a large chorioretinitic scar in the macula. The right eye has a small area of choroidal atrophy with pigment, nasal to the disc.

Katharine H. Chapman,
Secretary.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

June 8, 1948

LEUKEMIC INFILTRATION OF ORBIT

DR. J. WESLEY MCKINNEY reported the case of J. H., aged 5 months, who was referred for removal of a tumor of the right lower lid. The parents first noticed a redness and then a lump in the right lower lid which had progressively enlarged for one month. The child was otherwise apparently well. There was a firm mass involving most of the floor of the right orbit. The mass was not attached to the skin but attached to the periosteum. The lower lid was bulged outward and the eyeball displaced upward slightly. The globe was internally and externally normal. The other eye and orbit were normal. The baby was sent to a local pediatrician for general physical examination. He reported a white blood count of 105,000, with 98-percent lymphocytes, and severe anemia. He made the diagnosis of acute lymphatic leukemia. The child died within two weeks.

ECZEMATOUS KERATITIS

DR. J. WESLEY MCKINNEY reported the case of Dr. M. C., aged 32 years. She gave the history of having repeated ulcers of the left cornea for the past four years. For the past six months the eye had been slightly injected and irritated to such an extent that she was able to carry on her work with difficulty. She was a resident physician at the Cleveland Clinic and had had many examinations and had tried many forms of treatment. Physical examinations were negative except for a low basal metabolism rate for which she was taking 3 gr. of thyroid daily. She was allergic and had been found sensitive to many things. Desensitization with various allergens gave no relief. Among the medications tried were vitamins generally and locally, riboflavin injections intrave-

nously, adrenalin drops, holocaine and sulfathiazole ointments. She was finally advised to leave Cleveland.

When first seen here, the right eye was entirely normal. The epithelium of the left cornea showed widespread stippling of the cornea. Almost the entire corneal surface stained with fluorescein. There was only slight pericorneal injection and moderate blepharospasm. Corneal sensitivity was slightly reduced. With the use of a protective goggle, which allowed a minimum of air to reach the cornea, the condition cleared to some extent and the staining areas fluctuated in size from time to time. During this time all medication was stopped. The patient, however, was never free from discomfort.

It was finally decided to try X-ray therapy on the basis of the good results often obtained in eczema of lids. A total of 600 r. was given in 6 equal doses at 4-day intervals. There was a moderately severe conjunctival reaction to the X rays which lasted about four weeks. During this time the stippling of the corneal epithelium could still be seen but there was no staining. The patient reported being entirely comfortable. One month later the corneal epithelium was entirely clear and there was no discmofort. Two months later or four months after the X-ray therapy, there had been no return of symptoms and the cornea was entirely clear.

Central scotoma due to pituitary tumor

Dr. Ralph O. Rychener presented R. P. S., a man, aged 25 years, who, two months previously, had discovered a loss of central vision in the left eye. Various examinations were done in his own city and he was seen at the Veterans Bureau at Jackson, Mississippi, where removal of the left eye was advised because of intraocular sarcoma. Examination disclosed normal vision, visual field, and normal eye grounds in the right eye. Vision in the left eye was reduced to moving objects and the field was inconclusive, although central vision was definitely lost. The fundus of the eye was heavily tessellated and, at the periphery far above, there was a dense accumulation of choroidal pigment which was interpreted as a benign melanoma.

The patient was returned to his referring physician, Dr. J. C. Pegues of Greenwood, Mississippi, and was advised to have a course of treatment consisting of foreign protein and vasodilators, since the condition seemed to be a retrobulbar neuritis. At the completion of this treatment, there was no visual change and he was seen again in Jackson, Mississippi, where the advice of the removal of the eye was reiterated.

Upon the patient's return to Memphis the same diagnosis was made as on the previous visit and he was admitted to the Eye, Ear, Nose, and Throat Hospital for further foreign protein and vasodilator therapy. At the conclusion of this treatment, vision in the left eye returned to 6/60, J12. At this time the form and color fields disclosed a temporal hemianopia in the left eye and a relative hemianopia in the right eye. Diagnosis was then changed to that of a pituitary lesion. X-ray films disclosed an eroding pituitary tumor and on May 11th, Dr. Nick Gotten removed a chromophobic adenoma of this gland.

Visual improvement was almost immediate and, within two weeks, it had returned to 6/6, J1 in the left eye, with slight improvement in the visual field and a residual pallor of the temporal portion of the optic disc. X-ray therapy through four portals is completing the treatment.

Progressive enlargement of filtration bleb following iridencleisis

Dr. Philip Meriwether Lewis presented a white woman, aged 52 years, with an increasingly large corneal bleb following operation for glaucoma.

On August 16, 1945, the right eye began to pain and, when seen the following day, was stony hard with vision reduced to hand movements. Treatment with eserine, pro-

stigmin, and mecholyl failed to lower the pressure. Iridencleisis was performed, the scleral incision being made ab externo, approximately 1.5 mm. from the limbus. The iris prolapsed, was split meridionally, and both pillars incarcerated into the angles of the wound. Convalescence was uneventful, the pressure remaining around 20 mm. Hg (Schiøtz) for about a month. Vision returned only to 20/70, which was partially due to some early lens opacities. Six weeks after operation the tension was only 12 mm. Hg and the filtration bleb was quite prominent, but did not extend forward past the limbus. Vision was 20/70. The left eye was normal.

The patient was not seen for seven months (April, 1946) at which time vision and tension were the same, but the bleb was uncommonly large and extended slightly into the cornea. She was asked to return every few months, but did not come back for over a year (July, 1947). She then had an acute attack of glaucoma in her left eye. Reduction of pressure could not be obtained by strong miotics, so an iridencleisis was performed. The anterior chamber did not restore for 10 days and has remained shallow ever since. Vision could not be improved to better than 20/100 after operation and the gradual development of a cataract reduced it to 15/200.

The bleb of the left eye was larger than average, but small in comparison to that of the right. The bleb of the left eye now extended 1 mm. over the limbus into the cornea; that of the right extended 4.5 mm. downward toward the pupil—from the 10- to 2-o'clock positions on the limbus, a distance of 13 mm.—and it was more than 2 mm. thick. It formed a mass that was uncomfortable to the patient and disfiguring.

The tension could not be measured due to the bleb covering more than one third of the cornea. Tension of the left eye was 8 mm. Hg and that of the right eye seemed about the same to palpation. The patient was reluctant to undergo further surgery, but it was felt to be necessary, as the bleb was destroying the cornea, dissecting it somewhat in the manner of an aneurysm. Removal of the corneal portion of the bleb, curettage of the upper part of the cornea, and the drawing down of a conjunctival flap have been contemplated. Possibly cauterization of the upper cornea should be done to try to secure a firm cicatrix and to prevent a recurrence of the enormous corneal bleb.

Daniel F. Fisher,
Recorder for Eye Section.

OPHTHALMOLOGICAL SOCIETY OF MADRID

April 16, 1948

Fatty degeneration of cornea

Dr. Marín Amat read a paper on two cases of fatty degeneration of the cornea following herpes corneae, one traumatic, the other spontaneous.

In both cases there was a rapid infiltration of fat in the whole cornea, during a period of two to three weeks, with complete loss of vision. It thus appears that herpes corneae predisposes the eye to such fatty infiltration. The fact that one case was due to an accident while the patient was at work gives it great importance from a legal standpoint.

In connection with these cases, Dr. Marin Amat discussed the presence of fatty substances in the cornea as possibly being caused either by a simple accumulation of fat occurring normally in the circulation, or by the production in situ of lipoids caused by foreign substances stimulating the tissues. In exceptional cases it may be due to an excessive quantity of fat in the circulating blood.

The fatty substance is found in a very fine emulsion in the blood stream and in the cells, constituting an integral part of the protoplasm and participating in its activities, though to a minor extent, since it is continually destroyed by the intracellular

oxidase. But if the oxidation is deficient, the fine emulsion of neutral fat and of cholesterol condenses and gives the typical picture of fatty degeneration.

Thus this process can be caused by local, insufficient oxidation, by excessive cholesterol in the diet, or by a disturbance of lipoid metabolism. In accordance with these concepts, Dr. Marin Amat suggested applying locally substances which provide abundant oxygen to the corneal tissues.

Discussion. Dr. Mario Esteban said that Dr. Marin Amat's paper touched on a topic of great practical interest since little is known of the intimate mechanism of local and general metabolic disturbances which lead to the fatty degeneration of the cornea. The therapeutic suggestions are scientifically sound and should be investigated, especially as we are now powerless to stop these processes.

Dr. Marin Amat agreed with Dr. Mario Esteban that secondary fatty degeneration of the cornea is a relatively frequent complication, but he does not know of any published case of its following herpes corneae. In addition, herpes corneae is considered an occupational disease when it accompanies or follows ocular trauma. Moreover, it seems that the herpes virus tends to dispose the corneal tissue to fatty degeneration.

Retinal detachment diagnosed as retrobulbar neuritis

Dr. Carreras-Matas described a case of a woman patient whose only symptom was a central scotoma. The ophthalmoscope showed no alteration at the posterior pole of the fundus which could explain the scotoma, and it was diagnosed as retrobulbar neuritis. Several days later, the scotoma became denser and spread to the periphery. The ophthalmoscope showed a retinal detachment with a very minute tear in the centrocecal region.

Dr. Marin Amat said that a small retinal detachment may be confused in the beginning, because of the central scotoma, with retrobulbar optic neuritis, since the papillomacular bundle is involved in the neuritis. In the case described the papillomacular bundle was also involved by the slight edema and the subsequent detachment of the retina, which in the beginning could not have been detected.

The only difference which may be mentioned is that, in optic neuritis, the papillomacular bundle is affected in its course in the optic nerve and, in this case, in its course between the fovea and the papilla.

It is evident that the blue blindness which the detached zones of the retina show, which in turn is a form of hemeralopia, can also be explored by perimetry.

In the days when there was no operation for retinal detachment and the detachments became larger and more fixed, it was common observation that the patients would tell us when the retina became adherent. Although they could not see the color of the sky during the day, in the morning on opening the window they were enthused on seeing the beautiful, intense blue color of the sky. What happened was that, during the night, with the absorption of the subretinal edema, the retina became attached, although only for a transitory period.

John I. Pascal,
Translator.

AMERICAN JOURNAL OF OPHTHALMOLOGY

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THE JUNE MEETINGS

The American Ophthalmological Society held its 85th annual meeting at Hot Springs, Virginia, on June 2nd to 4th. The weather was perfect, the surroundings beautiful, comradeship evident, and the program highly satisfactory and instructive. There were 120 members and 29 guests in attendance, and 21 scientific papers were read and discussed. Most of these were meritorious and several were particularly noteworthy.

To mention briefly a few, Dunnington discussed late fistulization of operative wounds and described an operation by suture to close the fistula. Cordes described bilateral malignant ocular melanoma. Joy's paper on nevus flammeus associated with glaucoma added further evidence that increased capillary permeability may occur in the condition.

Scheie presented further evidence that goniotomy is efficient in congenital glaucoma. Reese contributed valuable material to the subject of spontaneous cysts of the ciliary body simulating neoplasms. Prof. G. B. Bietti, of Pavia, and Gundersen's paper on the use of para-amino-salicylic acid and streptomycin was well received.

One of the most interesting papers was

that by Dunphy on ocular conditions associated with idiopathic hyperlipemia. Dr. Bedell had some beautiful retinal Kodachromes, as usual, to illustrate his discussion of retinitis punctata albescens. Fink's contribution on the anatomic approach to surgery of the oblique muscles was convincing and valuable.

These papers and others similarly important produced lively discussions which often developed points of interest and instruction that were as significant, or at times more so, as many presented by the essayists.

The Howe Medal, which hereafter will be known as the Distinguished Service Medal of the society, was granted to Phillips Thygeson of California for his outstanding contributions to the field of ophthalmic bacteriology and virus diseases.

Parker Heath of Boston was elected the president, and John H. Dunnington of New York, the vice-president. The retiring president, Bernard Samuels, presented a beautiful chain and badge of office to the society, and graciously hung it about the neck of the incoming president.

The members of the society unanimously adopted a by-law to the constitution whereby a member found guilty of accepting rebates would forfeit his membership in the society. This is the first national ophthalmic organization to take this important and long-delayed stand.

Most of the members moved on to Philadelphia to attend the 18th scientific meeting of the Association for Research in Ophthalmology which was held on June 6th and 7th. At this meeting, 18 splendid papers of strictly research interest were given. Of these, the papers by Collins on "Experimental studies on sympathetic ophthalmia," and Smelser and von Sallmann on the "Correlation of microscopic and slitlamp examination of developing hereditary cataracts in mice," von Sallmann and Dillon on "Studies of the eye with radioiodine autographs," Kinsey on "A study of the possible conversion of dehydroascorbic acid to ascorbic acid in the aqueous humor," Koelle and Friedenwald on "The histochemical localization of cholinesterase," and Day on "Polysaccharides in ocular tissue," were particularly outstanding.

Wilson evaluated a radium-D applicator that has been widely sold and leased to ophthalmologists and pointed out that it has a low output and superficial penetration. An increase in the dosage of radium D to obtain adequate irradiation of deeper tissues would produce more damage superficially than radon or radium.

William F. Hughes, Jr., was elected the new trustee. Most of the 150 members registered stayed throughout the entire meeting, and one could not help but be impressed by the growth, enthusiasm, and great significance of this organization. There is no question that outstanding work in experimental ophthalmology is being accomplished by an increasing number of investigators who are native born and trained. The best of the new work appears to be done in the few "institute" type of organizations that are available in this country.

The Section on Ophthalmology of the American Medical Association held its annual session on June 8th to 10th at Atlantic City. There were 450 members of the section registered, although the audience at times was greater than this number.

M. Hayward Post, Jr., the chairman, in his address discussed the functions of the various national organizations in this country with particular reference to that of the section. He pointed out the key position in American ophthalmology occupied by the section and its importance to ophthalmologists.

A symposium on ocular injuries, put on the program at the request of the Surgeon General of the Army, followed Dr. Post's address. It was well received, and should be useful to the armed forces and to the general practitioners for future reference.

Ten scientific papers of excellent merit were given during the rest of the session. These were well discussed not only by the

official discussors but by members from the floor, as well. The subject of retrolental fibroplasia was reviewed by King and also by Heath who spoke on its pathologic aspects.

Among other noteworthy papers were those by Cowan and Klauder on "The frequency of the occurrence of cataract in atopic dermatitis," Bietti on "New trends in ciliary body surgery for the relief of glaucoma," and Sanders and Cutler on "General anesthesia for cataract surgery."

Ray A. Irvine of Los Angeles and Albert LeMoine, Sr., of Kansas City were elected chairman and vice chairman, respectively. Dr. Conrad Berens of New York was awarded the Scientific Medal of the section for his many important contributions to ophthalmology.

The section was represented by eight exhibits, all excellent, in the Scientific Exhibition of the parent organization. Of these, the exhibit by A. L. Kornzweig, of New York, on the pathology of the eye in old age received the Silver Medal of the association for Group II, and that of Peter Kronfeld, Roy Riser, and John Parker of Chicago, on glaucomatous excavations, a Certificate of Merit, Group II.

In making the busy round of these meetings, one is convinced that our science in this country is growing by leaps and bounds. Earnest and eager workers are increasing in numbers. The scientific and literary merit of their contributions is on a very high level of integrity and truth. The stimulation of hearing them is almost overwhelming and mildly depressing, especially to those of a slightly lazy nature. However, those who attended the meetings will, no doubt, return to their work determined to improve the quality of their activities and refreshed by their experience. Derrick Vail.

WILMER RESIDENT'S MEETING

The Wilmer Resident's Association held its eighth clinical meeting at The Johns Hopkins Hospital and University on April 6, 7, and 8, 1949. The association is composed of former senior residents of the Wilmer Ophthalmological Institute. Their annual clinical meeting provides an opportunity to review the work done in the institute during the past year, and to follow the activities of the former residents.

The program indicated the scope of the research which is in progress at the institute and was organized to present subjects of interest to the clinician and to the investigator as well. The influence of Dr. Alan C. Woods, the director, and of Dr. Jonas S. Friedenwald pervaded the meeting. Dr. Frederick Verhoeff was in constant attendance and took every available opportunity to present the "Boston" point of view in his inimitable discussion which delighted the group.

Dr. Woods presented what was in substance a symposium on uveitis. His three papers were entitled: "Classification and special symptomatology of uveitis," "The etiological diagnosis of uveitis," and "Treatment of uveitis." The granulomatous and nongranulomatous types of uveitis were clearly differentiated. Repeated attacks of nongranulomatous uveitis can produce vascular damage, thrombosis, and necrosis and, with subsequent repair, give a picture of plastic iritis which may superficially resemble granulomatous uveitis. Dr. Woods interprets this vascular damage as a manifestation of the Arthus phenomenon.

Granulomatous uveitis usually can be diagnosed and the etiology presumptively determined. An exception is in the case of chronic brucellosis. During the past year the armamentarium for indicating toxoplasmosis has been enhanced. The skin test of Frenkel, indicating sensitivity, and the methylene-blue dye test of Sabin show good correlation in evidence of the presence of neutralizing antibodies in the patient's blood. Sabin prefers the dye test to the rabbit test he formerly employed.

New possibilities for the treatment of uveitis are accumulating. Streptomycin,

promin and promizole in tuberculosis, aureomycin in brucellosis, penicillin in syphilis, and desensitization to bacteria causing the nongranulomatous type of the disease, are the outstanding examples. There is no question about the miraculous effect of penicillin on syphilitic uveitis. In other instances of the disease, penicillin is of no value and the promiscuous use of this antibiotic in cases not due to syphilis was censured. The final evaluations of streptomycin, promizole, and aureomycin await further clinical trial. The use of these and other new drugs and antibiotics was further discussed by Dr. Perrin Long in "Modern status of antibiotics."

In nongranulomatous uveitis due to sensitivity to streptococci, gonococci, and staphylococci, treatment by desensitization, particularly to specific strains of streptococci, is an approach to the problem that is being reemphasized. In some cases desensitization to specific organisms found in foci of infection is being carried on with encouraging results.

Putting time and effort into the study of each uveitis case, tracking down the etiology before instituting treatment, according to a rigid and carefully planned attack, as is done at the Wilmer Institute, is the only way the problems of this disease can ever be solved or judicious treatment be prescribed.

A fascinating paper by Dr. Malcolm W. Bick and Dr. Ronald Wood on "Heparin and ocular sensitivity" may prove to have a bearing on the problem of uveitis. They showed that heparinizing animals minimized or overcame induced states of hypersensitivity. Additional work along the lines of this investigation would seem to have great potentialities of application.

The perplexing and disheartening problems of uveitis are gradually being reduced, by such investigation as is being carried on at Wilmer, to more basic factors of tissue and humoral reactivity, and therapeutic measures are becoming accordingly more specific. The organization of uveitis services throughout the country, along the lines prescribed by Dr. Woods, would inestimably facilitate the care and cure of patients afflicted with this disabling disease. The goal is far from sight, but I predict that the tenacious and Herculean efforts of Alan C. Woods are setting the course and pattern for its realization.

"Sympathetic ophthalmia following intraocular surgery" was the subject of Dr. Howard A. Naquin of the resident staff. He analyzed 35 cases proved by pathologic examination. Many of these were operated on elsewhere than at Wilmer. In all cases there was evidence of incarceration of uveal pigment in the wound. The impression was that early enucleation of the activating eye has a favorable effect on the course of the disease. This is contrary to several previously reported investigations, particularly one which the author conducted in 1937. Further observations are warranted, as analyzing all cases in the literature where sympathetic ophthalmia is reported to have become apparent only after the exciting eye had been removed, and this eye revealing evidence of the specific infiltrate.

Dr. Jack S. Guyton, assistant director of the Wilmer Institute and associate professor of ophthalmology, read two papers on strabismus. The first, entitled "Surgical correction of horizontal strabismus: A new concept of the mechanics involved," emphasized the varying effect of the vertical muscles, particularly the obliques, on horizontal rotation as the eye is rotated laterally different degrees. The concept is based on mathematical formulas which heretofore have not been applied to the mechanics of ocular rotation. Application of Guyton's idea may lead to a more accurate estimation of the effects one may expect from recession, resection, and advancements. A simple mathematical formula to be used in surgery combined with a proper understanding of innervational factors is sorely needed if the results of strabismus surgery are ever to be uniformly predictable. His second paper, "Surgery of the horizontal recti: approach through a concealed conjunctival incision," demon-

strated a simple way to avoid unsightly scars without unnecessarily complicating the surgery.

Four papers dealt with the subject of glaucoma. The first of these was "Some ocular effects of a new anticholinesterase agent, tetraethyl pyrophosphate (T.E.P.P.)." Dr. William G. Marr, the present senior resident, presented evidence that this drug may cause detachment of the retina through extreme contraction of the ciliary muscle, and also that sensitivity reactions to this drug are so excessive as to preclude its clinical use at present.

Dr. Angus L. MacLean presented a method of "Corneoscleral trephine and intracapsular extraction in one procedure for primary glaucoma combined with cataract." Motion pictures were used to illustrate the procedure. The use of a Berens's punch in place of a trephine might possibly simplify the technique.

"The results of surgery in acute glaucoma" were analyzed by Dr. Herman K. Goldberg. Simple iridectomy was a satisfactory operation if the patient was being treated for a first attack of glaucoma. After a second attack, some type of iris-inclusion operation combined with separation of the iris at its base gave the best results. The last paper on glaucoma was "Changes in intraocular pressure following miotics," by Dr. Rufus C. Goodwin.

Two outstanding papers undoubtedly were inspired by the research of Friedenwald utilizing histochemical techniques for the study of physiology of the eye. The first, by Dr. Robert Day, "Polysaccharides in ocular tissue" dealt primarily with the distribution of polysaccharides and the effect of the enzymes, hyaluronidase and amylase, in depolymerizing these polysaccharides in the eye. The second, by Dr. George B. Koelle, was "Histochemical localization of choline esterase in ocular tissue."

Dr. Jonas Friedenwald's significant contribution was entitled "Inhibition of mitosis in the corneal epithelium by ionizing radiation." His experiments suggest that X rays inhibit cell division through chemical changes produced in the cells rather than through direct ionization of some component of the mitotic mechanism.

Dr. Roy O. Scholz concluded from his data on "Use of radioactive indicators for study of aqueous humor" that the aqueous is a secretion. He determined the concentration of radioactive sodium in the serum and the aqueous with the lids open and then with the lids closed.

Dr. Robert C. Laughlin, now associate professor of ophthalmology at the new medical school of the University of Washington, reported his interesting observation on "A new variety of corneal pigmentation" in workers in factories making poisonous gas. Approximately 50 percent of the employees in these factories showed what appeared as a Stahli's line in the cornea.

"New radon and radium-D applicators" were discussed by Dr. William F. Hughes, Jr. He has devised one in which small glass vials of radon gas are imbedded in beeswax in the tip of the applicator. The dosage can be varied by varying the number of vials used. Beta radiation, so applied, can obliterate vessels deep in the cornea, and the radiation is sufficiently strong so that it need be applied only for a very brief duration. Dr. Hughes explained that the radon-D applicators on the market give off a soft Beta radiation that can be used for surface-fire effect, but not effectively for destroying vessels deep in the cornea. The new applicator he described can be used for deep or superficial effect, and the time of application can be very short.

In "Cataract section following filtering operations," Dr. Russell T. Snip reviewed the results of such operations performed at the institute to determine which type of section affected the drainage least and produced fewest corneal changes. A corneal section away from the filtration bleb, rather than through it, was least detrimental to subsequent drainage in the eye. Sections above or

to one side, or below, in the cornea showed no appreciable difference in the incidence of subsequent dystrophic changes in the cornea.

Dr. Frank B. Walsh presented a masterful treatise on "The anatomy of neurologic defects in pituitary tumor." Diagrams and illustrations depicted clearly the anatomic basis for various signs and symptoms of pituitary disorders.

A thorough study of "The effect of retrobulbar alcohol injection on the eyes of experimental animals" was made by Dr. Walter Kornblueth, who was formerly at Wilmer and is now working at Stanford under the direction of Dr. Alfred E. Maumenee, the former Wilmer resident who is now professor of ophthalmology at Stanford Medical School. In the experimental animals, repair processes in the cornea were definitely inhibited by the retrobulbar injection of alcohol. The significance of this finding may not be as ominous as one might suppose, for clinical reports on patients reveal no alarming complications following such injections. However, this new experimental evidence should put the clinician on guard.

It was interesting to hear Dr. Walter H. Benedict discuss "Prognosis for vision in anoxia," because of the content of the paper and the manner of presentation, and, in addition, because of his resemblance to a certain well-known ophthalmologist from the Middle West who was an attentive listener.

From the department of physiological optics, Dr. Louise L. Sloan and Miss Lorraine Wollach reported on "Total color blindness."

In the field of electrophysiology Dr. Carlton C. Hunt, Dr. Stephen W. Kuffler, and Dr. Samuel Talbot demonstrated "New retinal recording methods from the intact eye." Action currents were recorded from single nerve elements.

A collection of fundus and external eye pictures were exhibited in the institute during the meetings. These drawings were made by Annette S. Burgess and represent the finest collection of fundus drawings in the country. Many hours could be profitably spent studying these pictures.

The outstanding single presentation at this meeting was that of Dr. William C. Owens and Dr. Ella Uhler Owens. The title was "Vitamin-E studies in relation to retrolental fibroplasia." These workers have already proved that retrolental fibroplasia is an acquired condition, first manifesting itself about two months after birth as dilatation of the retinal vessels with subsequent edema and exudate of the retina, then separation of the retina into peripheral folds that become organized behind the lens, finally forming a gray membrane. Because the condition is acquired, some factor in the care of prematures was suspected in the etiology. Because of the presumed poor assimilation of fats by the premature infant and because of the anemia and growth requirements, the accepted treatment in recent years has included high protein diet with high vitamin-A and iron intake. It has been shown that in some animals, particularly in chicks, high doses of vitamin A and iron inhibit the availability of vitamin E, resulting in a relative deficiency of E, leading to nutritional encephalomalacia.

No such clinical entity as vitamin-E deficiency has been recognized in humans, but the facts in experiments with chicks led the Drs. Owens to believe that deficiency in available vitamin E might be a factor in the development of retrolental fibroplasia.

Three groups of prematures were observed. The first group were treated in the usual way and the incidence of retrolental fibroplasia was the usual figure of 12 to 25 percent. A second group were given a new water-miscible vitamin-E preparation and less vitamin A and less iron than the first group. No instance of development of the condition was seen in this group. A third group, in whom the disease was just becoming apparent, were treated with addition of vitamin E and reduction in vitamin A and iron intake

and regression or cure of the disease resulted.

In discussing this paper Dr. Kinsey of the Howe Laboratory paid tribute to the accomplishments of the Drs. Owens as showing perspicacity of the highest order in the plan of investigation and the analysis of results, and he said that, in his opinion, those privileged to be present had witnessed an epochal contribution. It remains a mystery why the incidence of the condition is much higher in Boston, Baltimore, and Chicago than in some other cities, as for instance, Los Angeles, when the accepted modern treatment of prematures includes correspondingly high doses of vitamin A and of iron in all localities.

It is to be hoped that many of these fine papers will soon be published, for they are worthy of a nationwide audience.

S. Rodman Irvine.

BIFOCALS FOR JUVENILES

Not infrequent are misconceptions as to the care and significance of presbyopia and as to the prescription of bifocals. In infancy the healthy crystalline lens is so nearly fluid that its consistency presents no appreciable obstacle to the changes necessary in accommodation. If the eye is approximately emmetropic, all that is necessary for accurate vision of near objects is such an amount of activity of the ciliary muscle as will produce the proper increase of convexity of the crystalline lens. If the eye is made emmetropic by proper correction of hyperopia, myopia, or astigmatism, the same sort of physiologic need is satisfied by the same sort of action on the part of the ciliary muscle. Apart from uncorrected refractive error, this physiologic effort of accommodation normally causes no symptoms and is not harmful to the patient.

There used to be a rather general notion that the hyperopic patient possessed more, and the myopic patient less, than the normal capacity for accommodation. More exact study has shown this not to be the case. The myopic patient's accommodation, in the presence of an accurate distance correction, is normally as ample as that of the emmetropic or hyperopic patient under similar conditions of age and general vitality. Thus it is a mistake to assume that the properly corrected myopic child or adult is less equal to the task of reading, writing, or drawing than other properly corrected children.

Exact measurement and recording of the patient's accommodation is too generally neglected or carelessly undertaken. It should preferably be indicated in diopters on the basis of the least distance at which the patient can still see sharply (even with effort if necessary) the smallest detail of type or other fine test object which can be made out at such distance; the test being made either without or with correction, or with a presbyopic addition, according to the circumstances of the case.

Statements have been made as to the influence of convergence in relation to the myopic eye, but there is no adequate evidence as to the significance of any such relationship. Nor is there, in spite of all that has been said as to the effect of near work in the production of myopia, any proof that the mere exercise of accommodation, apart from the bearing of posture and other conditions upon the general health, is capable of damage to the young eye.

In the normal course of life the crystalline lens steadily becomes less fluid or elastic, and the near point of accommodation gradually recedes from the eye, until at last the eye is no longer capable of sufficient adjustment to provide for the ordinary range of close work, especially reading or sewing. In most eyes with proper distance correction this stage is reached somewhere around the age of 45 years.

It used to be rather customary to lay down a "rule of thumb" that the patient should be given (over and above his distance correction) about one diopter of plus spherical

addition at the age of 45 years, two diopters at 50, two and a half diopters at 55, and three diopters at 60 years. These figures are more or less excessive for most people, and were quite certainly dependent upon the fact that it was formerly much less usual to give patients adequate basic correction for hyperopia, so that they were regarded as being more presbyopic than they actually were. With proper distance correction, most people can manage very well with a 2.25 diopter near addition until well beyond the age of 60 years.

Every accurate ophthalmologist encounters from time to time a patient who has been given a bifocal addition unnecessarily. In the experience of the present writer, such cases have usually been those in which the basic hyperopia was inadequately corrected. More rare, to most of us, is the experience of seeing bifocals placed on young children. Now and then we have to do with an optician's or optometrist's prescription for a case of strabismus in which the distant correction has been grossly underestimated.

According to an ophthalmologist correspondent in the Middle West of the United States, there is in his vicinity a sort of "epidemic" of the furnishing of bifocals to cases of high myopia, or even sometimes to cases of only moderate myopia. This practice is seen, according to our correspondent's experience, among the respectable type of optometrist, and is explained as due to lectures given under a so-called "optometric extension program."

As examples of the practice are listed patients of ages ranging from 10 to 20 years, and with myopic errors running from minus 0.25 sphere through minus 1 or 2 diopters and up to 4 or 5 diopters. All the patients listed had excellent visual acuity, accommodation, and convergence. They were always grateful for withdrawal of the bifocal addition.

In defense of such a practice, the optometrist responsible was disposed to urge the frequent suggestion in older textbooks that the myopic correction prescribed for distance should be reduced for near work. It may be added that this older advise is more or less vaguely excused in some more recent publications.

The cause of marked differences in refraction, in families in which the hereditary factor is not clearly responsible, is far from being definitely understood, but there is good reason to suppose that it has something to do with an increased hereditary capacity for variation in the family or individual, especially in the period of active growth. It is now general practice to prescribe a full correction for this refractive anomaly, although of course with precise attention to astigmatic errors and inequality of the two eyes, as well as extreme accuracy in the placing of the lenses. The present writer has followed this procedure in very many cases of high or moderate myopia, in children usually healthy and physically vigorous, and has never seen cause to regret doing so. Nor, in adopting such a course, is it necessary to take the patient out of school. The rapid progression in amount of myopia frequently seen is almost certain to become much less marked at the close of the period of active growth.

One rather important point commonly overlooked is that the patient with an uncorrected low amount of myopia may suffer from eyestrain caused by habitual cramping of the lid muscles in the instinctive effort to sharpen vision.

Although, let us hope, usually done with a clear professional conscience, the ordering of bifocal glasses for myopic children is not only unnecessary but objectionable, and suggests in the prescriber a lack of scientific understanding of ocular physiology. The satisfaction subsequently created by withdrawal of the presbyopic element at the hands of an enlightened ophthalmologist must be somewhat disturbing to the reputation of the optometrist concerned.

W. H. Crisp.

OBITUARIES

ARTHUR N. ALLING
(1862-1949)

Dr. Arthur Nathaniel Alling was born in New Haven, Connecticut, July 1, 1862, of an old New England family. He prepared for college at the Hopkins Grammar School and received his B.A. degree from Yale in 1886, remaining for an extra year in the Sheffield Scientific School. In 1891, Dr. Alling was graduated from the College of Physicians and Surgeons, Columbia University. Having become interested in ophthalmology, he remained in New York for a year to work under the late Dr. Herman Knapp and began practice in New Haven, in 1893. In 1896, Dr. Alling became lecturer in ophthalmology at the Yale School of Medicine and, in 1902, was made clinical professor, which position he held until 1938.

A man of sterling character, keen mind, and ready wit, Dr. Alling was a skillful operator, a stimulating teacher, and enjoyed a large practice. He was a member of the New York Ophthalmological Society, the American Ophthalmological Society, and a Fellow of the New York Academy of Medicine.

Eugene M. Blake.

WARREN DOUGLAS HORNER
(1890-1948)

Dr. Warren Douglas Horner died on the night of October 22, 1948, in his home following a lingering illness. He was born September 11, 1890, at Klamath Falls, Oregon. After obtaining his preliminary education in Chico, California, he attended the University of California, where he was graduated in 1913. Three years later he received his M.D. degree from the university's medical school. Following this he served his internship at the San Francisco Hospital.

During the first world war, Dr. Horner served as a Navy lieutenant at base hospitals in Scotland and France. Upon discharge from the Navy he devoted one year to special study of the diseases of the eye, ear, nose, and throat at the University of Vienna, following which he entered private practice in 1923. From 1929 on, Dr. Horner limited his practice to the diseases of the eye and was appointed associate clinical professor of ophthalmology of the University of California and chief of ophthalmology of the University of California's Section of Ophthalmology at San Francisco Hospital.

Immediately after the attack on Pearl Harbor, Dr. Horner was recalled to active duty in the Navy with the rank of captain and was finally stationed at Oak Knoll Hospital, where he was retired in 1945.

He contributed numerous important articles to the ophthalmic literature, the most outstanding one being his thesis for the American Ophthalmological Society entitled, "A study of dinitrophenol and its relation to cataract formation."

Dr. Horner was honored by his colleagues on numerous occasions serving as chairman of the eye sections of the county and state societies. He was the first president of the San Francisco Ophthalmological Round Table, and was vice chairman of the Section on Ophthalmology of the American Medical Association at its centennial celebration in Atlantic City in 1947.

In addition to his appointment to various San Francisco hospitals, he was ophthalmic surgeon and consultant at the United States Marine Hospital of San Francisco.

Dr. Horner was a member of the San Francisco County Medical Society, the California Medical Association, the American Medical Association, the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, the Association for Research in Ophthalmology, the San Francisco Round Table, and the Pacific Coast Oto-Ophthalmological Society.

On June 28, 1919, he was married to Madge Clendenin. To this happy marriage with its beautiful home life were born a

daughter, Mrs. Harold A. Hyde, and a son, Douglas C. Horner.

Aside from his practice, Dr. Horner's chief interest was in his work with the students and residents. Persistence and attention to detail were primary virtues that he tried to instill in his associates. He was a good teacher, and gave freely of his time in this field. He was devoted to his work, and was an intelligent observer and a skillful operator.

Dr. Horner was tall, handsome, friendly, with a modest personality and a keen sense of humor, and was always ready with a clever story. His charm, graciousness, and love of people gained for him a host of friends within and outside his profession. To his friends he was known as "Bud," and because he was so real and so gracious he is remembered by thousands of friends, patients, and students.

Frederick C. Cordes.

BOOK REVIEWS

CORNEAL GRAFTS (keratoplasties) (Les Greffes de la Cornée). By L. Paufique, G. P. Sourdille, and G. Offret. Published under the auspices of the French Society of Ophthalmology. Paris, Masson et Cie, 1948. 359 pages, 135 figures, 20 plates, 4 of which are in color, bibliography. Price, not listed.

The authors have succeeded in giving us a well-illustrated, factual description of corneal grafting in all of its manifestations from history, instrumentation, and the various techniques to a discussion of the healing and its complications. The indications and contraindications for the operation are clearly set forth. It is gratifying to note the prominence given to American authors in the contributions to this field of ocular surgery.

An analysis of the results of the authors' partial nonperforating keratoplasty operations showed 71 anatomically favorable results out of 90 cases (75 percent) according to the criterion of Filatov, that is, the graft remained transparent, permitting one to see the details of the anterior chamber and the ocular fundus. There were eight poor results. These results are a little better than those obtained from keratectomies.

The book is well printed and is a worthy companion of those hitherto published by the same firm under the auspices of the French Society of Ophthalmology. Ophthalmologists who read French will get much out of it. It is hoped that an English edition may become available.

Derrick Vail.

DA BIOMICROSCOPIA ESTÈROSCÓPICA DO FUNDO DO ÔLHO DO CÃO NA VIGÊNCIA DA HIPERTENSÃO (Experimental stereoscopic Biomicroscopy of the Ocular Fundus of the Dog in the Course of Experimental Hypertension). By Cyro de Barros Rezende. Independently printed monograph, São Paulo, Brazil, 1948. Stiff paper covers, 167 pages, 28 plates, 21 in color, 7 in black and white.

This is the author's thesis presented to the Faculty of Medicine of the University of São Paulo in competition for the chair of professorship of clinical ophthalmology, "approved with distinction." Following to some extent the lines of Goldblatt's work on the same general topic, the author describes (in Portuguese) his modified method of slitlamp study of the ocular fundus of the experimental animals, with ample illustrations of the special apparatus employed, as well as description and illustration of the supplemental apparatus for measuring the arterial pressure of the dogs. Goldmann's contact lens was successfully applied to this study.

The beautiful color drawings, under the slitlamp, of the tapetum lucidum and nigrum in normal animals and in the course of the hypertensive studies were made by Professor Rezende's São Paulo colleague, Dr. A. G. Silva. There are also five excellent photomicrographs of retinal changes.

Interference with the circulation of the

kidney in the 24 experimental dogs was accomplished by clamping the retinal artery with Goldblatt's silver forceps.

The author states the following conclusions: Of all the ocular changes in benign experimental hypertension of the dog, the first and most constant is superficial edema of the retina. This is chiefly located in the tapetum nigrum and was most frequently evident in those retinal areas traversed by the large vessels. Even in the incipient edemas, the light bundle of the slitlamp showed slight elevation of the internal limiting membrane of the retina. As the edema became more intense, the retina lost its transparency and became grayish white. These appearances were confirmed in microscopic sections. In the benign experimental stage there was also accentuation of the median reflexes of the retinal arteries, with sheathing of the vessels and compression at the arteriovenous crossings. Changes encountered in malignant experimental hypertension included one instance of intense intraocular hemorrhage, white patches with fluffy margins, papillary edema, and marked lateral tortuosity of the retinal arteries. (List of 72 references.)

W. H. Crisp.

RÉPERCUSSIONS SUR L'ENFANT DES MALADIES INFECTIEUSES DE LA MÈRE PENDANT LA GROSSESSE. By F. Bamatter. Basel and New York. S. Karger, 1949. 60 pages, 24 figures. Price, 7.50 francs.

In this excellent monograph the author discusses, in general, the exogenous factor in the fetal dysgeneses and the placental transmission of maternal infections. A full-page table lists the bacteria, viruses, protozoans, and helminthes, the placental transmission of which has been demonstrated in man and in animals.

After a very brief statement of the known facts of the transmission of syphilis, tuberculosis, infectious icterus, paratyphus, and malaria, monographic treatment is accorded toxoplasmosis and the embryopathy of rubella.

A tabulation makes all the important clinical and pathologic data perspicuous. Excellent halftones illustrate the monograph, two of which are particularly illuminating. In one, the sites of localization of toxoplasmosis are indicated on a drawing of a fetus together with brief statements on mode of infection. In the other the tissues which may be injured by maternal rubella are indicated on a model of a 4-mm. (one-month) human fetus.

F. H. Haessler.

BULLETIN SOCIÉTÉ BELGE D'OPHTALMOLOGIE. Number 89, 1948.

In the minutes of the meeting of June 20, 1948, is a discussion of the eye signs preceding death. In speaking of retrobulbar injection, one author says that he uses a 5-cm. needle and injects 1.5 cc. of novocain-adrenalin (4 percent) solution.

Van Lint discusses the role of pressure on the globe in intracapsular cataract extraction and explains his technique which includes a conjunctival flap, keratome and scissors section, total iridectomy, and removal of the lens by suction, coupled with pressure from below toward the center of the globe with an Arruga expressor.

A case of total ossification of the choroid following an injury 60 years before is presented.

Of the two instruments discussed, one is for the removal of foreign bodies from the cornea and the other is a shadow-casting device to be used with the biomicroscope.

Among the case reports are a case of parenchymatous keratitis with keratoplasty resulting in a final vision of 0.1; two cases of Groenouw's disease of the cornea; and one of hypercalcemia and corneal calcifications.

Ophthalmoscopic manifestations of myasthenia gravis are discussed, and photographs

are shown of the patient before and after the injection of prostigmin.

Three cases of gargoylism are presented. Three cases of keratoconjunctivitis sicca are mentioned, with various treatments, including closure of the tear points, section of the sympathetic nerves, irradiation of the lacrimal gland, injections of pilocarpine and prostigmin, installation of gelatin drops of Gifford, vitamin-B_2 therapy, and male-hormone therapy.

There is a gonioscopic picture of a case of acute iridocyclitis. Two cases of acute glaucoma which followed the subconjunctival injection of adrenalin for the treatment of iritis are described. Treatment of four cases of optic neuritis are discussed. Intra-arachnoid injections of penicillin were given.

There is a discussion of therapeutic tissue implants in the treatment of retinitis pigmentosa. Among the ocular manifestations in one case of this disease were irregular distribution of the cutaneous pigment, light perception only, convergent strabismus, nystagmus, atrophic globes, seclusion of the pupil, miosis, and hypotony of each eye. It is stated that only 11 comparable cases have been reported in the literature. It is a congenital syndrome.

Two cases of retinal tears without detachment are discussed.

Bennett W. Muir.

TRANSACTIONS OF THE OPHTHALMOLOGICAL SOCIETY OF PARIS (and of the Ophthalmological Societies of the East, of Lyon, and of the West). Meeting of June, 1948, pp. 242-328; meeting of July, 1948, pp. 334-401.

Mme. Nada Matavulj reported her observations on the tissue therapy of Filatov. In spite of the short time of this study (3 years), it seems to be clear that the biogenic stimulation in the form of placenta, placental extract, aloes, and cod-liver oil gave unexpected improvement in otherwise hopeless cases.

François Pierre reported on three children with retrolental fibroplasia. He describes the clinical appearance and differential diagnosis and stresses the fact that this anomaly was described in France in 1883 by Vassaux.

Delthil, Halbron, and Navdin describe two cases of postsurgical aniridia which they do not consider to be as critical an accident as is generally believed.

Thurel doubts the importance of opticochiasmal arachnoiditis but considers the blocking of the pericerebral arachnoidal spaces of influence because it causes the resorption of air over the hemispheres and accumulation of cerebrospinal fluid and, with it, distention of the cisterna basalis.

Bonnet found that the fundus pictures of amaurotic idiocy in a child with craniofacial dysostosis suggested that this latter condition might be not only a malformation and premature closure of the facial sutures but also a degeneration of the cerebrum and the retina.

In the July issue is an interesting and concise paper on the heterophorias, as delivered by Suzanne Braun-Vallon at the November, 1948, meeting of the French Society of Ophthalmology. The fundamental principles and clinical applications are adequately presented in six chapters, with 11 figures, charts, and a bibliography.

Alice R. Deutsch.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Bodian, M. **An aid in detecting trachoma-like inclusion bodies in the conjunctiva.** Arch. Ophth. **40**:147-151, Aug., 1948.

The inclusion bodies are observed in the conjunctival scrapings in cases of trachoma, inclusion conjunctivitis, psittacosis and lymphogranuloma venereum. A red light obtained by using the E light red photographic filter (Wratten), series 23A, over the light source, increases the visibility of the bodies in slides stained by the Giemsa method. John C. Long.

Samuels, B. **Necrosis of intraocular tissues.** Arch. Ophth. **40**:101-120, Aug., 1948.

Noninflammatory necrosis is caused by circulatory disturbances and may follow iridodialysis, cyclodialysis and glaucoma. Inflammatory necrosis may be caused by pyogenic bacteria in the cornea, anterior chamber or vitreous. Necrosis caused by trauma and by neuropathy and degeneration of malignant melanomas is described in detail. Artificially induced necrosis may follow radiation therapy for retinoblastomas and diathermy treatment of retinal detachment. (10 figures.) John C. Long.

Weiss, C., Shevky, M. C., and Perry, I. H. **Experimental investigation of the pathogenicity of diphtheroids isolated from the human conjunctiva.** Arch. Ophth. **40**:23-38, July, 1948.

The first attempt to establish an etiologic relation between a diphtheroid and a disease of the eye occurred in 1879, when Italian investigators isolated Corynebacterium xerose during the course of an institutional epidemic of xerophthalmia. Later, in 1883, German workers demonstrated the presence of diphtheroids on the conjunctiva of patients with a nutritional deficiency but it was soon realized that these bacteria are usually present on normal and inflamed conjunctivas and other surfaces of the body. In general, corynebacteria are nonmotile, grampositive, aerobic, nonsporulating rods, which resemble Corynebacterium diphtheriae, and usually show uneven staining and variable morphologic changes during growth. When mucin, which occurs in the conjunctiva and other mucous membranes, is used as a vehicle in which to suspend certain bacteria, the

organisms may acquire an increase in virulence for experimental animals. Mucin functions as a protective capsule for the micro-organisms, decreases phagocytosis and intracellular digestion of bacteria and reduces the bacteriolytic power of serums.

Diphtheroids, resembling C. xerose, which were isolated from the human conjunctiva, grew well in a menstruum of mucin. When suspended in this medium and inoculated into the anterior chamber of the eyes of albino rabbits, cultures retained their viability in vivo for several days, while in suspensions in saline solution they were rapidly destroyed. Intraocular injection of diphtheroids suspended in saline solution produced moderate inflammation of the ciliary process in albino rabbits. When suspended in mucin, the reaction lasted longer, was much severer and was associated with acute keratitis.

It may be concluded that diphtheroids, which are almost constantly present on the normal and inflamed human conjunctiva, may be potential pathogens.

Ralph W. Danielson.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Bonavolontà, G. **Microspectroscopic observations on the living eye.** Ann. di ottal. e clin. ocul. **74**:339-349, May, 1948.

In making his microspectroscopic observations Bonavolontà used a Gullstrand slitlamp fitted with Abbe's spectral eyepiece and with an arc light in place of the usual Nitra lamp. Conjunctiva, cornea, aqueous, and vitreous have a uniform continuous chromatic spectrum, without distinguishing peculiarities, but may present the characteristic spectrum of oxyhemoglobin in the presence of circulating or extravasated blood. The spectrum of the normal crystalline lens is shortened toward the red end (it does not extend beyond 620 to 630 μμ, whereas the spectrum of the conjunctiva extends to 660 or 670 μμ) and is relatively dim toward the violet end. These characteristics of the lenticular spectrum disappear as the lens becomes cataractous, so that the spectrum of a mature cataract is normal as regards extension and brightness. Spectroscopy affords a means of differentiating between hemorrhagic and inflammatory exudates in the vitreous. Harry K. Messenger.

Brolin, S. E. **Spectrometric, photoelectric determination of the fluorescence of the eye lens. A quantitative and objective method for experimental investigations.** Acta ophth. **26**:395-411, 1948.

During certain pathologic conditions of the lens fluorescence undergoes significant changes, and it is possible that fluorescence may be a sensitive indicator of metabolic changes in the lens. The author describes a method of spectrometric determination of the intensity of lens fluorescence, which is accurate enough for experimental investigation. The fluorescent light from a lens preparation in a quartz tube is analyzed with a spectrograph and an electron multiplicator photocell. The data can be recorded objectively, and small changes in the fluorescence of the lens can be recognized spectrometrically. There is a difference in the intensity and spectral localization between the cortex and the nucleus.

Ray K. Daily.

Davson, H. **Some considerations on the salt content of fresh and old ox corneae.** Brit. J. Ophth. **33**:175-182, March, 1949.

The eyes from freshly killed oxen were examined, immediately and after 24 hours of ice storage, for sodium, potassium, chloride and water content of the cornea and of the aqueous humor. After 24 hours the cornea had an increase of water content, a slight decrease in sodium and a slight increase of potassium and chloride.

There was a corresponding decrease in the concentration of ions in the aqueous which suggests that the increased hydration represents a migration of aqueous into the cornea and not a mere osmotic drainage of water from this fluid.

Morris Kaplan.

Feldman, J. B. **Mydriatics.** Arch. Ophth. **41**:42-49, Jan., 1949.

This paper evaluates the clinical merit of the commonly available mydriatics as to such factors as safety, speed of action and return to normal pupillary size. In this study 12 mydriatics were checked. Ordinarily, they worked more efficiently on cloudy days and on patients with blue eyes. Homatropine hydrobromide, 1 percent, atropine methylnitrate, 1 percent, and dibutoline sulfate, 5 percent, dilated the pupil in the greatest number of patients. Dibutoline sulfate, 5 percent, gave definite cycloplegia in association with mydriasis in the greatest number of patients.

Paredrine hydrobromide was less potent but was quite satisfactory as a mydriatic in a number of cases. Glaucoma is indeed a rare complication of mydriasis. Even when a mydriatic has been mistakenly used in "pre-glaucoma" one will obviate disaster by keeping the patient until the pupil has contracted particularly since much more powerful miotics than 1-percent pilocarpine hydrochloride are available. Ralph W. Danielson.

Jaffe, N. S. **Cholinesterase in the aqueous of the eye.** Arch. Ophth. **40**:273-278, Sept., 1948.

Theoretically, there can be only faint traces of cholinesterase in the normal aqueous, since the enzyme is a protein. Aqueous from the eyes of 10 normal cats showed no cholinesterase, or insignificant traces of it. The first and second aqueous after paracentesis showed marked cholinesterase activity; the amount is proportionate to the plasma protein in the aqueous. Ralph W. Danielson.

Jaffe, N. S. **Practical application of the denervated iris.** Arch. Ophth. **40**:317-325, Sept., 1948.

The denervated iris of the cat is too sensitive an indicator of the epinephrine content of foreign solutions, but may be used as a very sensitive indicator of the increase in the epinephrine level of the blood. In the stage of excitement of ether anesthesia the output of the epinephrine in eight cats was so great that it remained for an hour. Ether is sympathomimetic. Barbiturates inhibit the production of epinephrine due to ether anesthesia. Ralph W. Danielson.

Lehrfeld, L., and Donnelly, E. J. **Contaminated ophthalmic ointments.** Arch. Ophth. **40**:39-45, July, 1948.

The unused tube of ointment was in most instances sterile, whereas the used tubes were frequently contaminated. The observations in this study call for a revaluation of the use of ophthalmic ointments in the eye after operation and of the use of ointments for ophthalmic conditions in general. The authors have no proof that infections actually occur from the use of contaminated ointments, nor that the organisms found in the contamination were pathogenic bacteria but even sulfathiazole ointment and penicillin ointment may become contaminated before the contents of the tube are exhausted.

Ralph W. Danielson.

von Sallmann, L. **Controversial points in penicillin therapy of ocular diseases.** Arch. Ophth. **39**:752-804, June, 1948.

This is a comprehensive report based largely on detailed experimental studies of the use of penicillin in the eye. The parenteral us of the drug is investigated from the standpoint of the passage of penicillin across blood aqueous barrier,

penicillin levels in ocular fluids after ligation of the renal vessels and the passage of penicillin into the ocular fluids in ocular inflammation. The surface application of penicillin by means of solutions, ointments, and iontophoresis is studied. Studies are described of the injection of the drug into the conjunctiva, anterior chamber, lens, and the vitreous.

The best results from the systemic administration of penicillin in severe ocular infections are obtained by the administration of massive doses in the acute phase, aided by the simultaneous local instillation of vasodilators in less violent inflammations. The continuance of penicillin activity was usually observed in the tears for eight hours after one instillation of drops or ointment. The rather involved factors concerned in iontophoresis are discussed. With observance of specifically outlined requirements, direct injection of penicillin into the vitreous can be considered at present the most reasonable treatment of infections of the posterior segment. The many points revealed by this searching investigation should be of great value in the practical application of penicillin therapy in eye infections.

John C. Long.

von Sallmann, L., and Moore, D. H. **Electrophoretic patterns of concentrated aqueous humor of rabbit, cattle and horse.** Arch. Ophth. **40**:279-284, Sept., 1948.

Information about the effects on the eye of the drugs, which are usually administered locally, when they are administered systemically in their usual dosage, is limited.

In experiments the authors found that morphine sulfate administered intramuscularly in doses ranging from 8 to 18 mg. produced pupillary constriction, increase in accommodative power and decrease in ocular tension in normal eyes. Morphine produced similar effects in glaucomatous eyes; in none of the 14 eyes studied did its use lead to an increase in intraocular pressure. In equivalent doses, atropine, given intramuscularly, produced much less pupillary dilation and weakening of accommodation than did scopolamine. Neostigmine, in a dose which produced definite increase in intestinal peristalsis, had no consistent ocular effects.

Ralph W. Danielson.

Schlosshardt, H., and Adam, W. **The penicillin content of the aqueous after intramuscular, intravenous and local penicillin.** Klin. Monatsbl. f. Augenh. **113**:333-342, 1948.

The results, which were all obtained in animal experiments, correspond to those found in the American literature. (References.) Max Hirschfelder.

Theodore, F. H. **Use of propionates in ophthalmology.** Arch. Ophth. **41**:83-94, Jan., 1949.

The purpose of this paper is to acquaint ophthalmologists with a group of physiologic antibiotics, which have hitherto not been used in treatment of ocular infections, and which have certain advantages over medicaments now in general use. The group consists of the lower fatty acids which occur in the human body. In this preliminary report the sodium salt of one of these fatty acids, sodium propionate, was selected for study. The lower fatty acids are nontoxic, physiologic antibiotics and fungicides. Sodium propionate, a fatty acid derivative, was used clinically in about 400 cases of conjunctivitis, blepharitis, and keratitis. It has proved efficacious and nonirritating, especially in treatment of chronic conditions. Ralph W. Danielson.

Woods, A. C., and Burky, E. L. **Studies in experimental ocular tuberculosis.** Arch. Ophth. **39**:471-490, April, 1948.

The authors report on the experimental

results in the therapy of ocular tuberculosis in immune-allergic rabbits using two sulphones, "promin" and "promizone," as the therapeutic agents. Both of these agents exert a deterrent action on the course of the ocular tuberculosis. The action becomes evident after the third week of treatment. The action of the two drugs is about the same.

Histologic evidences of tuberculous disease persist in the majority of the eyes of the treated animals. The lesions, however, are fewer and are less severe than in the control eyes. In transfer experiments with the extract of the uveal tract from the eyes of the seven treated animals only one positive result was obtained. All of the control eyes gave positive transfers. These results may be due either to a degradation of the virulence of the organisms, allowing the resistance of the host to become effective, or to a direct bactericidal action. John C. Long.

Woods, A. C., and Burky, E. L. **Studies in experimental ocular tuberculosis.** Arch. Ophth. **40**:1-13, July, 1948.

In a previous study of the effect of treatment with two sulfone compounds, "promin" and "promizole," on experimental ocular tuberculosis in the immune-allergic rabbit, it was not clear whether the deterrent action of the sulfone compounds noted was due to a bactericidal action or to degradation or attenuation of the virulence of the organisms, thus allowing the resistance of the host to become more effective. The experiments here reported were undertaken to clarify this point. "Promin" and "promizole" used in the same dose as that which had a pronounced deterrent effect on ocular tuberculosis in the immune-allergic rabbit were only slightly deterrent on ocular tuberculosis in animals that had not been sensitized. These drugs probably have a limited bactericidal effect on the tubercle bacillus. Ralph W. Danielson.

Woods, A. C., and Wood, R. M. **Studies in experimental ocular tuberculosis: XII. Effect of streptomycin and "promizole" on experimental ocular tuberculosis in the immune-allergic rabbit.** Arch. Ophth. **40**: 413-432, Oct., 1948.

Streptomycin exerted a deterrent action on the course of ocular tuberculosis produced by inoculation of the eyes of immune-allergic rabbits. Despite the absence of clinical evidence of activity, histologic examination of the eyes showed minimal to moderate activity and transmission experiments and cultures showed that the tubercle bacillus was still alive in some of the eyes. A combination of streptomycin and "promizole" produced a more dramatic response. After four weeks of combined treatment, the diseased eyes were clinically and histologically inactive and transmission experiments were negative in more of the animals. Streptomycin has a definite bacteriostatic and a partial bactericidal action on the tubercle bacillus in ocular tuberculosis. The combination of streptomycin and "promizole" is somewhat greater than would be expected from a summation of their individual actions. John C. Long.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Auerswald, W. **The area effect in scotopic vision.** Ophthalmologica **117**:104-109, Feb., 1949.

For threshold stimuli there is a fairly simple mathematical relationship between intensity and duration of the stimulus as long as the portion of stimulated retina is kept small. Stimulation of larger retinal areas introduces a new factor, the so-called area effect. The occurrence or non-occurrence of the area effect seems to depend more upon the location than upon the total number of the stimulated retinal elements. Peter C. Kronfeld.

Balcet, A. **Myopia from the manufacture of woolen goods.** Rassegna ital. d'ottal., **17**:379-383, Nov.-Dec., 1948.

In a group of forty-two women workers, ranging from 16 to 19 years of age, only nine were emmetropic. The others had two to five diopters of myopia. The writer ascribes the myopia to the nature and conditions of their work, which is very exacting, is done at close range and in poor light. E. M. Blake.

Berger, Curt. **Some experiments on the width of symbols as determinant of legibility.** Acta ophth. **26**:517-569, 1948.

The purpose of this study was to determine the effect of a change in the width of symbols on their legibility. An important interaction takes place between all factors affecting legibility, such as form, width and height. This study is a part of a long-range statistical program which will emphasize the effect of interaction between the factors.

The graphic data show that the increased width of the letters results in an increased legibility which is explained as a purely retinal function, namely the resolution threshold of the fovea centralis. (19 graphs.) Ray K. Daily.

Best, F. **Embryologic considerations in the origin of the lateral inversion of the optic pathways in the brain.** Klin. Monatsbl. f. Augenh. **113**:234-246, 1948.

Best provides a critical review and an extension of the theories of Cajal.

Max Hirschfelder.

Bottoni, Angelo. **The light sense, and the various methods of measuring it in clinical practice.** Ann. di ottal. e clin. ocul. **74**:396-420, June, 1948.

This article is a monograph on the light sense and its measurement and is illustrated with diagrams of various adaptometers and other devices. Bottoni finds adaptometry clinically useful in the detection and measurement of vitamin A deficiency, in studying the role of vitamin A in community and racial health, and in the detection and treatment of night blindness in night workers.

Harry K. Messenger.

Casanovas, José. **Chalazion and astigmatism.** Arch. Soc. oftal. hispano-am. **9**: 23-27, Jan., 1949.

Transient astigmatism may be caused by a chalazion, and glasses prescribed before its extirpation may be unsuitable afterwards. Ray K. Daily.

ten Doesschate, J. **The relationship between the extrafoveal scotopic threshold and the distribution of retinal rods.** Ophthalmologica **117**:110-115, Feb., 1949.

This is another quantitative study of the relationship between visual function and the number of visual elements per mm.^{2} of stimulated retina. The histologic basis for most of the recent work in this field was furnished by Østerberg who reported the results of careful visual cell counts in the eye of a 16-year-old boy. Crozier and Holway compared these results with their own determinations of the extrafoveal absolute scotopic threshold of the light sense and found no simple relation between the two sets of data. Now ten Doesschate compares Østerberg's rod counts in the horizontal retinal meridian with the scotopic light threshold determinations of Stiles and Crawford and finds good agreement between the histologic and the visual data. The existing minor discrepancies can easily be explained by individual variations and other causes. These results strongly support the theory —already firmly established—that the cones and rods form the anatomical substrata of the photopic and scotopic mechanism, respectively. Peter C. Kronfeld.

Hardy, L. H., Rand, G., and Rittler, M. C. **Incidence of defective color vision among psychotic patients.** Arch. Ophth. **40**:121-133, Aug., 1948.

A high incidence of defective color vision in psychotic patients has been reported but the authors found that the incidence of color blindness in the 235 psychotic patients tested with pseudo-isochromatic plates was not significantly higher than that in the normal population.

John C. Long.

Henderson, J. W. **Cotton sutures in ophthalmic surgery.** Arch. Ophth. **39**:545-548, April, 1948.

Cotton fiber is an inert material with a natural twist which can be tied into small, stabile knots with a high tensile strength. It incites early healing with little undesirable tissue response. These characteristics are features that might be considered in the possible use of cotton as a suture material in ophthalmic surgery. The author illustrates the use of cotton sutures in a considerable variety of ophthalmic operations. John C. Long.

Kinsey, V. E. **Spectral transmission of the eye to ultraviolet radiations.** Arch. Ophth. **39**:508-513, April, 1948.

The ultraviolet absorption spectrums of various components of the albino rabbit eye have been measured. The limit of transmission for the whole eye is approximately 330 millimicrons; for the lens, 310, and for the aqueous and the vitreous and cornea, separately, approximately 280 millimicrons. In the corneal epithelium the chief absorbing element is nucleoprotein; its limit of transmission is less than 230 millimicrons. The amount of radiant energy from the sun to which the eye would have to be exposed before minimal damage occurs in the lens is calculated to be about three times the dose necessary to produce minimal damage to the cornea. So little ultraviolet radiation reaches the retina that damage would be extremely unlikely.

John C. Long.

Kugelberg, I. **On the effectiveness of tabulæ pseudoisochromaticæ B. K.** Acta ophth. **26**:429-437, 1948.

This is an evaluation of pseudoisochromatic tables published in Sweden. Color plates are of little value in differentiating protanomolous and deuteranomolous vision of moderate severity, and complicated figures may cause difficulties for persons with normal color vision.

Ray K. Daily.

Macnie, J. P. **Clinical aniseikonia.** Arch. Ophth. **40**:326-331, Sept., 1948.

A study was made of the 1,027 patients examined for aniseikonia at the Institute of Ophthalmology of the Presbyterian Hospital, New York, between December, 1936, and December, 1941. Aniseikonia was demonstrable in 527 and 463 received glasses. Of the latter, 365 (79 percent) were relieved of all or a part of their symptoms, and 98 (21 percent) were not benefited. In 1946, from five to ten years later, a questionnaire revealed that of 304 patients, one half were benefited. No symptoms are characteristic of aniseikonia, but symptoms associated with use of the eyes not relieved by the usual ophthalmic therapeutic measures may be due to it. The presence of aniseikonia cannot be anticipated on the basis of anisometropia or the magnitude of the refractive error. Nor is it possible to anticipate whether the correction of aniseikonia will aid the patient. Ralph W. Danielson.

Nolasco, J. B., and Rodil, D. **Responses to the Ishihara test for color perception.** Arch. Ophth. **41**:20-23, Jan., 1949.

Approximately 2,000 male and female Filipino students were tested with the Ishihara color plates. The incidence of defective color vision in men was 4.3 percent; in women 0.3 or 0.2 percent, depending on which criterion was used.

Ralph W. Danielson.

Pascal, J. I. **The "incident neutral" point in retinoscopy.** Arch. Ophth. **39**: 550-551, April, 1948.

The point where the emergent rays from the patient's eye meet is ordinarily spoken of as the neutral point. There is another neutral point which concerns the incident light and may be called the "incident neutral" point. This point is obtained when one uses a concave mirror and the incident light is made to focus in the plane of the patient's pupil. There will then be neutrality of motion irrespective of any refractive error the patient may have. John C. Long.

Pascal, J. I. **Real significance of centering a contact lens.** Arch. Ophth. **39**:514-516, April, 1948.

The prevailing method among physicians when centering a contact lens has been to center the cornea of the lens with the cornea of the eye. To get the best optical effect of the contact lens its cornea should be centered with the crystalline lens. Objectively this is impossible, but subjectively it can be done by observing the effect on vision of slight displacement of the position of the contact lens. Theoretical considerations of the variations in optic axis produced by contact lenses are discussed. John C. Long.

Paton, R. T. **Eye ointment dispenser.** Arch. Ophth. **39**:549-550, April, 1948.

A metal container is described into which the standard ointment tube is inserted. Only the tip of the sterilizable container comes in contact with the eye. (1 figure) John C. Long.

Rios Sasiain, Manuel. **The extrafoveal sensitivity of the retina.** Arch. Soc. oftal. hispano-am. **9**:147-151, Feb., 1949.

Physiologic nocturnal myopia was utilized for determination of the most sensitive parafoveal area. With a black disk 9 cm. in diameter on a white background, with a controlled illumination, and regulated extrafoveal fixation it was found that the area of greatest scotopic visual acuity is 15 degrees from the fovea. (2 graphs.) Ray K. Daily.

Rocco, Alfredo. **Practical questions on color vision.** Arq brasil. de oftal. **12**:10-28, 1949.

A review in Portuguese of tests and theories, with bibliography.

W. H. Crisp.

Rubino, A., and Pereyra, L. **The eye and the diencephalon.** Riv. oto-neuro-oftal. **23**:221-226, July-Aug., 1948.

The dark adaptation curve of congenitally amblyopic eyes were compared with similar curves of the normal eye of the same subject. The results confirm the conclusion reached by previous authors that light sensitivity undergoes no changes in amblyopic eyes. Moreover in amblyopic eyes it undergoes the same changes in "day-night rhythm" as normal eyes. These results corroborate the hypothesis that the lesion causing the amblyopia is in the intracerebral optic ways.

Melchiore Lombardo.

Toledo, Celso. **Refractometry based on measurement of circles of diffusion.** Arq. brasil. de oftal. **12**:1-9, 1949.

The author describes a test based upon the apparent distance of objects of complementary colors, 5 m. or more from the patient, seen through two pinholes of complementary colors, separated from one another by a distance less than the diameter of the pupil. (7 black and white figures, references.) W. H. Crisp.

Valente, Adolpho. **Accommodation and some of its aspects in refraction.** Rev. brasil oftal. **7**:143-162, March, 1949.

The author's talk is clinically illustrated by three case reports, one on devergence insufficiency with a spasm of accom-

modation and convergence, one of spasm of accommodation with convergence insufficiency, and one of anisometropia with compound myopic astigmatism.

W. H. Crisp.

5

DIAGNOSIS AND THERAPY

Benton, C. D., Jr., and Heyman, A. **Treatment of ocular syphilis with penicillin.** Arch. Ophth. **40**:302-310, Sept., 1948.

During the past three years the authors have used penicillin in the treatment of 39 patients with various manifestations of syphilis of the eye. Penicillin produced little or no immediate response in interstitial keratitis but the opacity cleared and the inflammation subsided in three to five months. Good or excellent vision resulted in 80 percent of the eyes treated in the first attack, but penicillin did not always prevent keratitis in the second eye. Penicillin treatment of six patients with acute iritis associated with secondary syphilis healed immediately and vision became normal. Papilledema associated with syphilitic meningitis responded well to penicillin therapy. In four of eight patients with primary optic atrophy the process seemed arrested 11 to 24 months after treatment. Combination with fever therapy is advised for optic atrophy. Ralph W. Danielson.

Cimbal O. **The methods of penicillin therapy in eye diseases.** Klin. Monatsbl. f. Augenh. **113**:342-353, 1948.

The local use of penicillin in diseases of the eye is far superior to general, as only weak concentrations of penicillin reach the corneal tissue and the interior of the eye after intramuscular or intravenous injection except in pyogenous infections of the adnexa. Conjunctivitis may be treated with drops but corneal infections need repeated 15-minute applications of penicillin, 50,000 to 200,000 units per cc., by means of a cotton applicator. For penicillin therapy of the interior of the eye a cotton tampon with a few drops of 100,000 units per cc. penicillin solution is introduced into the lower conjunctival fornix. This method is preferred when pure crystalline penicillin is not available for subconjunctival injections. (References.) Max Hirschfelder.

Fedrizzi, G., and Ferri, L. **Soft-ray roentgentherapy of superficial eye diseases.** Boll. d'ocul. **27**:697-705, Nov., 1948.

Plesioroentgenotherapy is recommended for practically any inflammatory lesion on the surface of the eye. If the equipment is not available, one may use any roentgen apparatus and apply 30 to 40 r per session until the total dose is 180 to 320 r. A short review of 31 corneal and scleral lesions shows that in some cases a satisfactory result can be obtained.

K. W. Ascher.

Fiore, Tito. **Pentothal anesthesia in ocular surgery.** Ann. di ottal. e clin. ocul. **74**:381-395, June, 1948.

Pentothal anesthesia is preferable to inhalation anesthesia and is particularly indicated in the treatment of injuries to the globe, in acute glaucoma, in operations on the adnexa, and in enucleation and exenteration and other destructive operations. Administered by rectum it is the anesthesia of choice in operations on children.

Harry K. Messenger.

Gardilčić, A. **The use of rat tail tendons as suture material in ophthalmology.** Ophthalmologica **117**:115-126, Feb., 1949.

As reported earlier by K. Pischel, rat tail tendons make satisfactory absorbable material for corneoscleral sutures. The author describes a convenient method of obtaining and preparing the tendon and reports experimental studies with various suture materials imbedded intracorneally

in rabbits. Rat tail tendon is better tolerated than catgut, silk or human hair.

Peter C. Kronfeld.

Gifford, H. **Motor block of extraocular muscles by deep orbital injection.** Arch. Ophth. **41**:5-19, Jan., 1949.

In order to get more complete motor anesthesia and lower vitreous pressure, the author has (after considerable experimental work and cadaver dissections) devised an altered technique in retrobulbar injections which he describes in detail. Using 2 cc. of 2 or 4-percent procaine hydrochloride, with 0.4 cc. of epinephrine hydrochloride to the ounce (29 cc.), he was able to produce a better than 60-percent motor block in 74 percent of 158 cases. Ralph W. Danielson.

Grancini, L. E. **Experimental anaphylaxis with normal placenta and with Filatow's preparation.** Rassegna ital. d'ottal. **17**:357-366, Nov.-Dec., 1948.

The writer transplanted placenta under the conjunctiva 30 times in man for various intra-ocular pathological processes according to Filatow's method. Animal experiments demonstrated that the Filatow placental tissue was not anaphylactic when implanted in the peritoneal cavity or in the orbit. Fresh placental tissue, under the same conditions, produced an anaphylactic reaction. Filatow's tissue is not toxic and does not produce anaphylaxis in animals sensitized by fresh placental tissue. The conclusion is reached that the therapeutic results are no better with Filatow's material than with fresh placenta and therefore the latter is preferable. Eugene M. Blake.

Lindsay-Rea, R. **Eyeball rotating forceps.** Brit. J. Ophth. **33**:193, March, 1949.

A locking forceps, whose toothed blades terminate in sharply bent arms, which is used for rotating the eyeball is described.

Morris Kaplan.

McMackin, J. V. **Fixation light.** Arch. Ophth. **40**:351-352, Sept., 1948.

In order to simplify the screen test the author has designed a light that can be displayed in the cardinal direction of gaze.

Ralph W. Danielson.

Morano, M., and Franchi, B. **Ocular therapy by low-voltage irradiation at close distances.** Boll. d'ocul. **27**:625-638, Oct., 1948.

Low-voltage irradiation at close distance in corneal diseases was beneficial and the authors used the same treatment for diseases of the inner coats. They used a Goral-Siama apparatus, Chaoul type, and a 3-mm. celluloid plate to protect the surrounding ocular tissues from secondary radiation. Usually the dose was 160 to 165 r for uveal, and 190 to 195 r for deeper lesions, using 55 kV, 5 mA, focal distance 5, and the celluloid filter on alternating days for 5 or 6 applications. (3 figures.)

K. W. Ascher.

Perera, C. A. **A simple appositional suture for use in operations for cataract.** Arch. Ophth. **40**:347-350, Sept., 1948.

Perera describes his method of placing appositional sutures after keratome incision. Ralph W. Danielson.

Raiford, M. B. **Dispenser for adhesive tape.** Arch. Ophth. **39**:816-817, June, 1948.

A plastic dispenser is described.

John C. Long.

Ruedemann, A. D. **Beta radiation therapy.** Arch. Ophth. **41**:1-4, Jan., 1949.

The author discusses the technique of use of the beta ray application in conjunctival and corneal abnormalities and dermatologic growths of the lid. There is a twofold hazard in the use of radiation therapy: to the patient by overdosage, faulty application or severe reaction, and to the ophthalmologist. Means of avoidance are described.

Ralph W. Danielson.

Urrets Zavalía, Alberto. **The action of diamino-diphenyl-sulfone on the ocular complications of leprosy.** Arch. Soc. oftal hispano-am. **9**:160-175, Feb., 1949.

This is a tabulated report of the application of this drug in 25 patients with leprosy. The drug prevents the severe uveal complications of leprosy but mild transient iritis may occur during the course of treatment. It is incapable of checking the evolution of slowly progressive ocular leprous lesions. When the anterior uvea is not involved the drug hastens the absorbtion of corneal infiltrates and brings about the disappearance of the thickened corneal nerves. It cannot arrest the progress of the lesion when the iris and ciliary body are affected. Ray K. Daily.

6

OCULAR MOTILITY

Berg, J. L. **Management of vertical heterophoria of paretic origin.** South. M. J. **42**:220-224, March, 1949.

Bielschowsky's four types of vertical heterophoria are reviewed. The symptoms of vertical heterophoria vary from a mild drowsiness after one hour of close work to a marked blepharospasm with a chronic blepharoconjunctivitis. The amount of prism prescribed depends on the patient's occupation and upon the amount of variation in the findings between the heterophoria for near and for distance.

H. C. Weinberg.

Casari, G. F. **Clinicopathogenic importance of heterophoria.** Rassegna ital. d'ottal. **17**:389-399, Nov.-Dec., 1948.

Twenty cases of heterophoria of various grades were studied and treated with vasodilators and antispasmodic drugs (nicotine amide and chloride of benzilimidazoline). The results were practically nil and not as good as those reported by Angius who administered acetylcholine. Orthoptic exercises were more effective. Eugene M. Blake.

Costenbader, F., Bair, D., and McPhail, A. **Vision in strabismus.** Arch. Ophth. **40**: 438-453, Oct., 1948.

Conclusions are presented based on a study of 407 cases of squint. From the considerable data given, several generalizations may be made. Visual acuity can be estimated in the infant by determining the ability in foveal fixation. The greater frequency of amblyopia in patients with strabismus can be predicted from a history of "constant" or "monocular" deviation. While the average age of onset of squint may not be significant, the average duration of squint before treatment is most significant in the production of amblyopia. Amblyopia is more frequent with convergent than with divergent squint, and with mechanical convergent than with accommodative convergent squint, and is least frequent with alternating squint. The incidence of amblyopia increases directly with the degree of hypermetropia or the degree of anisometropia present. Total occlusion is the treatment of choice for amblyopia ex anopsia. Helpful suggestions for carrying out the occlusion are given.

John C. Long.

Gailey, Watson. **The crosseyed child—a social as well as a medical problem.** New Orleans M. and S. J. **101**:387-389. Feb., 1949.

The psychologic problem of the child with squint is of great importance. The child withdraws from social contacts, and becomes nervous and capricious.

R. Grunfeld.

Noronha, Marianna. **Some considerations as to anomalous retinal correspondence.** Rev. brasil. oftal. **7**:131-141, March, 1949.

After a general review of the subject

the author proposes there should be established in Brazil a commission, with instructions to present at the next Brazilian ophthalmological congress suggestions for uniform nomenclature of strabismus, available for scientific work and statistics. (7 figures.) W. H. Crisp.

Swan, K. D. **The blindspot syndrome.** Arch. Ophth. **40**:371-388, Oct., 1948.

The blindspot syndrome, of which 102 cases have been observed, consists of periodic diplopia, a concomitant esotropia of 10 to 20 degrees in which the physiologic blindspot of the deviating eye overlies the area of regard, and normal retinal correspondence. Treatment consists of correction of the refractive error, development of ample fusion by orthoptic training and slight overcorrection of the esotropia by surgical measures. John C. Long.

7

CONJUNCTIVA, CORNEA, SCLERA

Barroquer Moner, J. I. **Technic of penetrating keratoplasty.** Arch. Soc. oftal hispano-am. **9**:152-159, Feb., 1949.

The principal feature of Barraquer's method is the direct suture of the transplant to the cornea of the host. He uses eserine to constrict the pupil, believing that a small pupil permits of better centration of the trephine and prevents anterior synechia. (5 figures.)

Ray K. Daily.

Bunge, E. **Keratoconjunctivitis epidemica found in lower Silesia in 1944.** Klin. Monatsbl. f. Augenh. **113**:369-376, 1948.

The author observed a form of keratitis superficialis punctata which probably belongs to the form of epidemic keratoconjunctivitis observed in 1938.

Max Hirschfelder.

Citterio, M. **Two cases of metastatic furunculiform episcleritis.** Boll. d'ocul. **27**: 666-672, Oct., 1948.

Two mature women developed metastatic episcleritis a few weeks after infected intragluteal injections. Similar cases have been reported; their significance is the evidence of a generalized metastatic disease, possibly dangerous to the life of the patient. K. W. Ascher.

Corrado, A., and Toselli, C. **Metastatic scleral abscess.** Rassegna ital. d'ottal. **17**: 367-378, Nov.-Dec., 1948.

All recorded instances of metastatic scleral abscess since 1881 are reviewed and abstracted. A 76-year-old man had a generalized furunculosis that had originated in the scrotum. These lesions cleared but several months later there was pain in the left eye and swelling of the lids, considerable exophthalmos, reduction of motility, chemosis with loss of corneal epithelium and a turbid aqueous. The fundus reflex was lacking. Spontaneous rupture of the abscess occurred and eventually the eye was enucleated. Six months later the other eye had an avascular keratitis punctata with absence of pupillary reaction and furuncles of the external auditory canal with staphylococcus. Eugene M. Blake.

Ellman, P., and Weber, F. P. **Sjögren's syndrome.** Brit. M. J. pp. 304-305, Feb. 19, 1949.

A case of Sjögren's syndrome is presented in which the respiratory tract was involved. Irwin E. Gaynon.

Friede, Reinhard. **Medical and surgical therapy of serpigenous corneal ulcer.** Acta ophth. **26**:509-516, 1948.

The author applies alcoholic solutions of bacteriostatics directly to the ulcer after preliminary cocainization; the alcohol causes a flow of fluid until it is neutralized in the tissues, and the stasis about the ulcer is thus relieved. The application is made eight times daily, and

the bacteriostatic is rubbed into and under the ulcer; the necrotic tissue is wiped away. The scars following this treatment are thinner than those after caustics or cautery.

When medical treatment does not stop the progress of the ulcer, the author removes the entire area down to Descemet's membrane with a large trephine. If the floor of the trephine area is clean it may be grafted with a piece of clear cornea; if it is infected the regeneration of the parenchyma is left to itself. When the entire cornea is involved a total keratoplasty will prevent an iris prolapse, with adherent leucoma, staphyloma, and secondary glaucoma. It results in an opaque cornea, which may be dealt with subsequently by further keratoplasties.

Ray K. Daily.

Goldsmith, J. **Deep keratitis associated with atypical lichen planus.** Arch. Ophth. **40**:138-146, Aug., 1948.

Deep keratitis in a soldier with atypical lichen planus associated with alopecia areata, poliosis, vitiligo and hyper-pigmentation is described. The cornea was not vascularized and the keratitis finally underwent complete regression. The patient had received 55 grams of quinacrine ("atabrine") for malaria. It is thought that quinacrine, as a sensitizing agent, and toxic products of the destructive cutaneous lesions, were probably responsible for the development of the keratitis. John C. Long.

Grom, Edward. **Limbal intrascleral cyst.** Ann. d'ocul. **182**:52-56, Jan., 1949.

A six-year-old child, struck in the left eye by an arrow six years before, developed a cyst at the limbus. The eye was enucleated because of degeneration and pain. The cyst contained conjunctival epithelium and the walls were formed by sclera which had undergone hyalin degeneration. Chas. A. Bahn.

Rados, A. **Conical cornea and mongolism.** Arch. Ophth. **40**:454-478, Oct., 1948.

The author has recently observed two cases of bilateral conical cornea associated with mongolism. The clinical characteristics of mongolism and the reported associated eye abnormalities are described in detail and the extensive literature on the etiology of mongolism is reviewed. The etiology of conical cornea is discussed from the standpoint of its association with vernal conjunctivitis, and with various endocrine and hereditary factors. It is possible that conical cornea may arise from endocrine abnormalities, particularly when associated with mongolism. John C. Long.

Redl, T. **Treatment of ophthalmia neonatorum at the University Eye Clinic in Vienna.** Klin. Monatsbl. f. Augenh. **113**: 353-362, 1948.

The results of penicillin therapy in 37 cases are tabulated. The solutions ranged from 250 to 1,000 units per cc. and drops were used at 10-minute intervals from 3 to 12 hours. In all but five a culture was negative within four to ten hours. The author had the impression that a 1,000-unit solution is irrating and that a 500-unit solution used for 12 hours promises the best results with the least chance of recurrences. Max Hirschfelder.

Roggenkaemper. **Treatment of keratoconjunctivitis nummularis with ether.** Klin. Monatsbl. f. Augenh. **113**:377-378, 1948.

The author drops 4 to 6 drops of ether on the anesthetized cornea in keratoconjunctivitis epidemica. Atropine and bandage follows this procedure. The eyes became well in six days, one third of them without any scars. Max Hirschfelder.

Rosen, E. **Interstitial keratitis and vestibuloauditory symptoms following**

vaccination. Arch. Ophth. **41**:24-31, Jan., 1949.

The report by Cogan of the syndrome of nonsyphilitic interstitial keratitis and vestibuloauditory symptoms of unknown etiology has stimulated interest in this subject. Because of a suggested etiologic factor in the case reported, this paper is submitted as an introductory study of postvaccinial encephalitis. In a review of Cogan's four cases Mogan and Baumgartner's case, and the one reported present case, certain clinical features stand out rather prominently: 1. repeated negative Wassermann and allied serologic reactions in patient and parents; 2. bilateral ocular and auditory symptoms; 3. occurrence in young adults; 4. chronicity, with multiple unpredictable recurrences; 5. synchronous onset of ocular and of aural symptoms; 6. mildness of ocular symptoms; 7. aggravation of symptoms by changes in the weather; 8. striking variation in symptoms; 9. characteristic yellowish-white corneal opacities, tending toward vascularization; 10. evidence of iritis (cells in the anterior chamber and keratic precipitates); 11. leukocytosis and suggestive eosinophilia; 12. bilateral nerve deafness; and 13. constant condition of the cornea. Ralph W. Danielson.

Silva, A. I. da. **Familial dystrophy of the cornea.** Rev. brasil. oftal. **7**:113-129, March, 1949.

Four members of the same family were affected, two brothers and two sisters from 16 to 30 years of age. Five other children were not involved. The affection had first appeared at the ages of 7 and 5 years in the men, and in the women at 6 months. The periphery of the cornea was spared in every case. All four had extremely low vision. A small corneal biopsy in one showed epithelium in process of keratinization, irregular in thickness and generally atrophic. Bowman's membrane was absent and replaced by amorphous hyaline structure. There had been inflammatory exacerbations. The author regards the cases as representing an atypical form of macular degeneration of the cornea. (9 figures, including 6 photomicrographs.) W. H. Crisp.

Stansbury, F. C. **Lattice type of hereditary corneal degeneration.** Arch. Ophth. **40**:189-217, Aug., 1948.

The lattice type of corneal dystrophy is a chronic, slowly progressive, familial disease of the cornea, inherited dominantly. It begins in the first decade of life, and usually in the first few months. It is characterized in youth by frequent recurrent erosions of the corneal epithelium, associated with severe photophobia, lacrimation and mild conjunctivitis. In adult life the recurrent attacks become less frequent and milder, but there is progressive loss of vision. The lesion is always bilateral. A detailed clinical and histologic description of the lesion is given. The etiology is unknown. The literature and classification of corneal dystrophies are reviewed. Five cases of lattice type degeneration are reported in detail. Keratoplasty was performed on one eye in each of three patients with favorable results in two. There is no assurance that the degeneration will not invade the transplanted tissue in time.

John C. Long.

Watson-Williams, E. **Blue sclerotics, fragilitas osseum and deafness.** Bristol Med-Chir. J. **65**:82-86, Autumn, 1948.

The triad that consists of blue sclerotics, deafness and fragilitas osseum occurred in a family of four. Inheritance is dominant. The maternal grandmother was married twice. Her descendants by both husbands show the triad. (2 figures, 2 tables). Irwin E. Gaynon.

8
UVEA, SYMPATHETIC DISEASE, AQUEOUS

Braley, A. E. **The etiology and treatment of uveitis.** J. Iowa St. M. Soc. **39**: 57-60, Feb., 1949.

The diagnosis and treatment of uveitis are uncertain and discouraging. Observations on supposed tuberculous uveitis are contradictory. Tubercle bacilli have been found in normal retinal tissue, and the adjacent choroid and ciliary body showed extensive inflammation but no bacilli. Tissues inoculated into guinea pigs produced no reaction. In other organs tubercle bacilli are almost always associated with the lesion. Other causes are also discussed. Rather extensive experimental studies of bacterial allergy and attempts to isolate a filterable virus are being reported. In the author's laboratory intracerebral inoculations of anterior chamber fluid from about 20 eyes with uveitis were made into mice and into fluid tissue cultures but revealed no infectious agent. Ground tissue from the iris and ciliary body of five eyes enucleated because of uveitis was incubated and inoculated intracerebrally into mice, guinea pigs, and rabbits. No organism was found. Typhoid vaccine is still the treatment of choice. Antibiotics and antihistaminics have been ineffective. Bacterial allergy is probably the most important cause.

F. M. Crage.

Casanovas, Francisco. **Sarcoma of the ciliary body.** Arch. Soc. oftal hispano-am. **9**:184-188, Feb., 1949.

The interesting features were an iridodialysis produced by the tumor, which is typical of tumors of the ciliary body and is exceptional in choroidal tumors, the formation of pseudocysts in the ciliary procesess, and inflammatory phenomena. The latter are important in differentiating tumor and tuberculoma of the ciliary body. (2 figures.) Ray K. Daily.

Marín Amat, M., and Marín Enciso, M. **Leucosarcoma of the iris treated by extirpation.** Arch. Soc. oftal. hispano-am. **9**:180-183, Feb., 1949.

A 46-year-old woman had a small symptomless growth in the iris of the left eye for 12 years. Then hemorrhages appeared which were slight and transitory for a few months and finally filled the eyeball and obscured vision. The growth, a leucosarcoma, was extirpated. (2 figures.)

Ray K. Daily.

Rome, S., and Koff, R. **Preplacement of air in cyclodialysis.** Arch. Ophth. **40**:134-137, Aug., 1948.

Rome and Koff have found it technically easier and safer to inject the air into the chamber through a tiny corneal incision before the cyclodialysis is done than after the operation is completed. The preplaced air cushion prevents hemorrhage instead of arresting bleeding which has already commenced. The deepened chamber facilitates the dialysis and the angle and cleft can be seen throughout the entire procedure. John C. Long.

Tower, Paul. **Congenital grouped pigmentation of the retina.** Arch. Ophth. **39**: 536-541, April, 1948.

Two cases of congenital grouped pigmentation of the retina are presented with fundus photographs. The typical arrangement of the deposits of pigment in a triangular sector of the inferior portion of the retina suggests that this anomaly may have a cause similar to that of coloboma of the choroid. John C. Long.

9
GLAUCOMA AND OCULAR TENSION

Arruga, H. **An adrenalin preparation similar to Glaukosan.** Arch. Soc. oftal. hispano-am. **8**:1206, Dec., 1948.

Arruga calls attention to a preparation under the name of Sol-inhal.

Ray K. Daily.

Barkan, O. **Technic of goniotomy for congenital glaucoma.** Arch. Ophth. **41**:65-82, Jan., 1949.

Goniotomy is an operation for stripping or peeling embryonic tissue from the wall of the angle. If the cornea is clear, the operation is performed under direct vision with the aid of a prismatic contact glass specially devised for this purpose. If cloudiness of the cornea prevents the use of the glass, the operation is performed without it.

Goniotomy, which at the time of this writing had been performed on 76 eyes with infantile glaucoma, preserved useful vision in most cases. In 66 eyes pressure was normalized and vision maintained or restored over periods ranging from one to ten years. In 10 eyes the operation was unsuccessful. When combined with early diagnosis, it provided excellent visual results. The mode of action of goniotomy is discussed. The advantages, disadvantages, indications, contraindications and technic of goniotomy are described. The importance of early diagnosis and prompt operation is stressed.

Ralph W. Danielson.

Campos, R. **A combined antiglaucomatous operation, cyclodialysis with basal iridectomy.** Boll. d'ocul. **27**:689-696, Nov., 1948.

To combat chronic simple and chronic "inflammatory" glaucoma, Campos modified Wheeler's suggestion to combine cyclodialysis and iridectomy by using a basal iridectomy instead of the complete iris excision which is too mutilating for common use. The peripheral iridectomy should be located at the site of the cyclodialysis. The latter is performed in the 12-o'clock region. A conjunctival flap is dissected to expose the upper rectus muscle and a bridle suture is introduced. Five mm. behind the limbus, a 2 to 3-mm. incision is made in the sclera, parallel to the limbus. A "first step" of cyclodialysis follows, separating the ciliary body only 3 mm. from the scleral incision without reaching the pectinate ligament. After that, another incision is made 1.5 mm. behind the limbus with a keratome, and a peripheral iridectomy is performed. The "second step" of the cyclodialysis follows. An Elschnig spatula is introduced into the anterior chamber through the first incision, and moved from the temporal edge of the coloboma to the 12-o'clock position, then from the nasal edge of the coloboma to the same meridional position. The spatula is withdrawn, re-introduced and the maneuver is repeated on the nasal side. (9 figures.) K. W. Ascher.

Crisp, W. H. **Early development of the filtration operations for glaucoma.** Ophth. ibero am. **10**:211-221 (English); 222-230 (Portuguese), 1948. See Am. J. Ophth., 31:3, p. 277, 1948. W. H. Crisp.

Dellaporta, A. **Results of antiglaucomatous iridencleisis, and the value of early operation.** Acta ophth. **26**:413-428, 1948.

This is a detailed analysis of the results of 224 cases operated by iridencleisis. It is just as effective as Elliott's operation without harboring the threat of late infection, and it is more effective than cyclodialysis. In noninflammatory glaucoma early operation was outstandingly more effective in preserving the visual field and reducing the tension. In late operations the number of failures was twice as large. These results are in conformity with those published by others. The earlier the operation is performed the better is the chance for normalization of tension and preservation of the visual fields. Of the 224 eyes, only 6 percent had free fistulization. The lining of the wound with protective uveal tissue accounts for the rarity of late infection after iridencleisis. (6 tables.) Ray K. Daily.

Diaz-Domínguez, D. **Glaucoma and excavation of the papilla.** Arch. Soc. oftal. hispano-am. **8**:1219-1242, Dec., 1948.

This is an exhaustive review of the literature and a report of several cases of glaucoma without hypertension, in which careful investigation revealed transitory phases of hypertension. The size and depth of the excavation of the papilla have no relation to the degree of ocular hypertension. The absence of excavation in prolonged hypertension is accounted for by a diminished rigidity of the ocular walls; on the other hand, disproportional increase in the depth of the excavation is related to an increased rigidity of the ocular walls, and a vascular sclerosis in the circle of Heller. In many reported cases of glaucoma without hypertension transitory or intermittent hypertension were probably overlooked. Glaucoma is a disease of age, and the adjective "senile" may be applied to it legitimately. (5 visual fields.) Ray K. Daily.

Esente, I. **Treatment of glaucoma with intravenously injected procaine.** Ophthalmologica **117**:147-160, March, 1949.

Two to three hours after the intravenous injection of 10 to 20 cc. of a 1 to 2-percent aqueous solution of procaine the ocular tension of glaucomatous eyes drops. Acute glaucomas respond better than chronic ones. The injections must be given slowly but may be repeated two to three times daily. The possible mode of action of this medication is discussed.

Peter C. Kronfeld.

Friedenwald, J., Kronfeld, P., Dunnington, J. H., Chandler, P. A., and Vail, D. **Symposium: Primary Glaucoma.** Tr. Am. Acad. Ophth. pp. 169-237, Jan.-Feb., 1949.

Glaucoma may be primary, secondary, and congenital, and primary glaucoma may be chronic simple, acute congestive, chronic congestive, and absolute. Practically all congestive cases are of the narrow chamber angle type. Vasomotor disturbances alone are not considered causative until the chamber angle has become at least partially obstructed. Primary normal and wide chamber glaucoma is usually of the chronic noncongestive or simple type. In some cases sclerosis of the afferent arterioles of Schlemm's canal was observed. Primary wide angle glaucomas may include the low tension group. Sudden lowering of intraocular pressure apparently results in the inadequate filling of Schlemm's canal with blood. Hypotony from penetrating surgery may cause ciliary edema and change a wide into a narrow angle glaucoma.

Kronfeld, P. **Diagnosis.** Pp. 175-185.

The classification of glaucoma as to chamber angle simplifies the early diagnosis of primary types. The early diagnosis of the narrow angle groups is usually not difficult. Most congestive cases belong in this group. The biomicroscope, gonioscope and dark room test are valuable. The early stages of wide angle glaucomas are usually more difficult to recognize, and a routine procedure is outlined which will reduce diagnostic failure to a minimum. Perimetric findings and low tension glaucomas are briefly discussed.

Dunnington, J. H. **The surgical treatment of primary glaucoma.** pp. 213-224.

Surgical intervention should be promptly considered in narrow angle glaucomas, unless adequately controlled by miotics and hygienic treatment. Operations should, if possible, be performed between acute episodes. In narrow angle glaucomas scleroiridectomy is usually contraindicated after 48 hours; then iridencleisis is usually the operation of choice. The trephine and other forms of sclerectomy are more traumatizing and therefore of more limited value. Paracentesis is of no permanent value. In chronic glaucomas the age of the patient,

tension, visual acuity and fields, combined with structural ocular findings, determine the choice of iris inclusion or sclerectomy operation. In chronic narrow angle glaucomas obliteration of the angle and peripheral synechia are bad prognostic signs. Sclerectomy types of operation are usually less satisfactory than iridencleisis if extensive peripheral synechiae exist. In chronic wide angle glaucomas cyclodialysis and iridencleisis are usually preferable in less advanced cases and trephine or other forms of sclerectomy in the more advanced. Goniotomy is of greatest value in infantile glaucoma.

Chandler, P. A. **Complications of surgery.** pp. 224-231.

The causes of operative failures as well as the methods of prevention and their correction are briefly discussed. Failure in trephine operations may result from scar tissue closure in congested eyes, high tensions with subsequent lens dislocations and friable conjunctiva. Meticulous attention to details of the procedure are essential. Simple iridencleisis may not be sufficient in far advanced glaucomas, especially if the iris is atrophic.

Cyclodialysis should not be employed if the anterior chamber is very shallow, the tension high or if extensive peripheral anterior synechiae are present. Cyclodialysis should include one-third to one-fourth of the circumference.

Vail, D. **Review, summary and conclusions.** pp. 232-237.

The importance of classification of primary glaucoma by the type of angle is briefly discussed and illustrated. Repeated attacks of acute narrow angle glaucoma lay the foundation for synechiae which increase with each attack. Operations on narrow angle glaucomas tend to produce analogues of acute attacks. Trabecular pigment may be of no importance. Low tension glaucoma must be differentiated from pseudoglaucoma with cavernous atrophy. Gonioscopic examination of potential glaucoma is advised. The early diagnosis of wide angle glaucomas may be exceedingly difficult and the five steps mentioned by Dr. Kronfeld are recommended. The importance of a synergistic combination of miotics of the different types is stressed. The authors of this symposium are in accord concerning the basic concept of the formation and drainage of the aqueous; the importance of recognizing narrow and wide angle glaucoma; the use of medical treatment especially for wide angle types only as long as it is demonstrably effective; and a careful choice of operation and thorough development of the operative techniques.

Chas. A. Bahn.

Lehrfeld, L. **Medical versus surgical treatment of glaucoma.** Arch. Ophth. **40:** 332-340, Sept., 1948.

This paper is a plea for conservatism. There are innumerable cases of acute glaucoma in which operation has been performed and the tension reduced, but the visual acuity has been reduced also. The author does not have record of a single case of acute glaucoma in which a surgical procedure has not resulted in some loss of vision.

Ralph W. Danielson.

Leopold, I. H., and McDonald, P. R. **Di-isopropyl fluorophosphate (DFP) in treatment of glaucoma.** Arch. Ophth. **40:** 176-188, Aug., 1948.

Di-isopropyl fluorophosphate has a prolonged miotic effect that results from an irreversible inactivation of cholinesterase. Its action is inhibited by the previous use of physostigmine or neostigmine. DFP successfully lowered ocular tension in 208 of 380 glaucomatous eyes. It was effective in concentrations ranging from 0.01 to 0.1-percent. Preparations of the drug in peanut oil were more effective than similar concentrations in liquid petrolatum. DFP lowered ocular tension successfully

in eyes with chronic glaucoma, acute glaucoma, buphthalmos, aphakic glaucoma, and glaucoma secondary to uveitis and exfoliating lens capsule, although other miotics had previously failed. The best results were seen in eyes with aphakic glaucoma. Decidedly fewer instillations of DFP were required to maintain a satisfactory ocular tension in eyes in which the tension was also controlled by other miotics. Disadvantages in its use include painful ciliary spasm, local sensitivity, and acquired resistance to its action. In six eyes a rise in tension followed its instillation. Retinal detachments have been reported to follow its use in patients with high myopia. John C. Long.

Marín Amat, M. **The true role of fistulating operations in the treatment of glaucoma.** Arch. Soc. oftal. hispano-am. **8**: 1249-1254, Dec., 1948.

The normal optic and metabolic functions of the eye are maintained by a neuro-vegetative intraocular center of regulation, represented by Muller's cells in the choroid, iris and ciliary body. All disturbances in the intraocular circulation, vascular or lymphatic, are compensated through this center, even if they originate extraocularly in the cortex, thalamus, medulla or hypothalamus. The primary cause of ocular hyptertension is an increase in the fluid content of the eye. Because of the compressibility of the venæ vorticosæ and Schlemm's canal, the hypertension impedes and may abolish the elimination of intraocular fluids. The interference with drainage is secondary to an overproduction of fluid. Antiglaucomatous operations should aim to decrease this fluid content of the eye by an attack on the regulating cells, situated at the root of the iris and in the ciliary body; they should in effect be arteriectomies or sympathectomies and diminish ciliary activity. Most effective are broad iridectomies and cyclodiathermies. External operations on the sympathetic ganglia are ineffective. Fistulating operations are effective only to the extent in which they act on the primary factor of overproduction of fluid, through the concurrent iridectomy. Cyclodialysis acts by destruction of the ciliary body and not by providing drainage through the suprachoroidal space. Diathermy-coagulation of the ciliary body acts directly on the nerve cells that regulate intraocular circulation. Marín Amat is emphatic in the opinion that fistulating operations are indicated only in cases in which surgical interventions aiming at the removal of the primary cause, such as iridectomies and cyclodiathermies are inadequate.

Ray K. Daily.

Moreu, Angel. **The role of the trigeminus in the regulation of ocular tension.** Arch. Soc. oftal. hispano-am. **8**:1193-1206, Dec., 1948.

Experimental data on dogs are presented to support the conclusion that excitation of the first branch of the trigeminal gives rise to an intraocular discharge of histamin, which through vasodilatation, increases the blood content of the uveal sponge, and raises the ocular tension. No modifications in tension were produced by the stimulation of the corneal plexus, but they appeared rapidly after irritation of the iris and ciliary body. The irritation was produced by a fine sharp needle introduced into the anterior chamber angle under gonioscopic observation, which punctured the iris or ciliary body; irritation of the iris was followed by a hyperemia of the ciliary vessels, a rise in tension, miosis, hyperemia of the iris and ciliary body demonstrable gonioscopically, and an increase of cells in the aqueous. Hyperemia of the ciliary vessels followed irritation of the cornea and conjunctiva. These experiments were repeated after modifying the neurovegetative ocular tone with mimetic and lytic

drugs. Parasympathomimetic drugs increased the effect of trigeminal stimulation and parasympatholytics were without effect. Sympathomimetics abolished the reaction of the trigeminal, and resection of the sympathetic and sympatholytic drugs exaggerated it. Acute rises in tension similar to acute glaucoma could be produced by blocking the vorticose veins concurrently with stimulation of the trigeminal. The experimental data are applied to the explanation of the pathogenesis of secondary hypertension in acute iridocyclitis. The rise in tension caused by an irritation of the trigeminus is not compensated because of the vasomotor paralysis, slowing of the circulation, and the obstruction in the venous and aqueous drainage incident to the disease. (6 graphs.)

Ray K. Daily.

Pallarés, J. **Surprisingly favorable results of cyclodialysis in advanced cases of chronic glaucoma.** Arch. Soc. oftal. hispano-am. **8**:1244-1248, Dec., 1948.

The author's enthusiasm for cyclodialysis is based on three cases in which it was followed by a marked reduction in tension and an improvement in very narrow visual fields. (3 visual fields, 1 graph.)

Ray K. Daily.

Posner, A., and Schlossman, A. **Syndrome of unilateral recurrent attacks of glaucoma with cyclitic symptoms.** Arch. Ophth. **39**:517-535, April, 1948.

Nine cases, forming a homogeneous group which represents a type of glaucoma intermediate between primary and secondary glaucoma, are described. The disease is unilateral. In three there was some heterochromia, and in each instance the lighter-colored eye was involved. Symptoms may be absent or there may be slight discomfort, colored halos or blurring of vision. Ocular hypertension may appear a day or so before, or simultaneously with, cells in the aqueous. Within the next 24 hours postcorneal deposits are seen. Episodes may occur with varying frequency and without any apparent cause. It is proposed to call this condition a syndrome of glaucomatocyclitic crises. A tentative hypothesis is advanced which relates this syndrome to a disturbance of the autonomic nervous system. A classification of glaucoma associated with iridocyclitis is presented.

John C. Long.

Rubino, A. and Pereyra, L. **The eye and the diencephalon. V. The "luminous sensibility" in simple chronic glaucoma.** Riv. oto-neuro-oftal. **23**:227-236, July-Aug., 1948.

There is a constant reduction of luminous sensibility in primary glaucoma which does not seem related to the changes of vision, visual field or tension but is attributed to a lesion of the diencephalic centers and the thalamus. (6 figures, references.) Melchiore Lombardo.

Rubino, A. and Esente, I. **The eye and the diencephalon, VI. Glycemic curve and "photo-glycemic reflex in glaucomatous patients.** Riv. oto-neuro-oftal. **23**:237-243, July-Aug., 1948.

Ten patients with simple chronic glaucoma showed glycemic curves that suggested changes of the diencephalic centers and also an abnormal "photo-glycemic" reflex. These observations on carbohydrate metabolism support the new concept of the relation between the diencephalon and glaucoma.

Melchiore Lombardo.

Stocker, F. W., Holt, L. B., and Clower, J. W. **Clinical experiments with new ways of influencing intraocular tension.** Arch. Ophth. **40**:46-55, July, 1948.

The influence of the "rice diet" introduced by Kempner for the treatment of hypertensive vascular disease on the ocu-

lar tension of nonglaucomatous patients was studied. All patients showed a striking and persistent reduction of ocular tension which did not seem to be directly dependent on the reduction of the blood pressure. It is believed that a relative depletion of chlorine and sodium ions in the tissues, including those of the eye, might be the underlying factor.

Ralph W. Danielson.

Vidal, F., Brodsky, M., and Travi, O. C. **The physiopathology of simple chronic glaucoma.** Ophth. ibero-am. **10**:107, 1948 (Spanish) 122, 1948 (English).

The nature and influence of three stages are discussed in detail. The exact interpretation of each developmental type permits us to avoid in future the gloomy prognosis. W. H. Crisp.

Weekers, R., and Humblet, M. **Angioscotoma and neuroscotoma of chronic glaucoma.** Acta ophth. **26**:455-467, 1948.

Every patient with early glaucoma was examined exhaustively on the perimeter and on the Bjerrum screen to demonstrate the earliest field changes in chronic glaucoma. Goldman had pointed out that increase in size of the angioscotoma is produced not by an increase in the size of the retinal vessels but by a functional inadequacy in the retinal nerve elements due to a vascular disturbance. The earliest symptom of glaucoma is a change in the physiologic angioscotoma, which goes through the three stages: increase in size, fusion, and increase in density. At the poles of the blind spot where the angioscotomas are close to one another they coalesce as they increase in size. The phenomenon is often interpreted as enlargement of the blind spot. These early changes are reversible. An increase in the density of the angioscotoma is a sign of a graver circulatory disturbance and indicates an irreversible lesion of the nerve elements. An angioscotoma with increasing density soon becomes a neuroscotoma. A neuroscotoma is easy to outline, and the examination should determine its position, extent, density, margins, and progress. They are arcuate in form, and extend nasally when they reach the nasal raphe. The peripheral extension of the arcuate scotoma accounts for the peripheral constriction of the nasal field. Extension in the temporal field constricts it until only a small centrocecal island of vision is left. The early visual loss of glaucoma is demonstrable only through patient and systematic perimetric and campimetric studies, with small test objects, and variable illumination. The diagnosis of these changes is important in that they form the indications for surgical intervention at a time when it can still be effective. (8 figures.) Ray K. Daily.

10

CRYSTALLINE LENS

Gaines, S. R. **The role of retrobulbar neuritis in the management of senile cataracts.** New Orleans M. and S. J. **101**: 390-391, Feb., 1949.

Many old people have markedly reduced vision although they have no lens changes. The visual loss is due to retrobulbar neuritis on an arteriosclerotic basis. The cavernous atrophy of the nerve head in "soft glaucoma" may be caused by pressure of the sclerosed internal carotid artery on the optic nerve. One may have arteriosclerotic optic atrophy without cupping of the disc caused by total or partial obliteration of small vessels. The atrophy may be diffuse or patchy, causing generalized depression of the visual field, sector defects, or central scotoma. Patients with lens opacities not dense enough to explain the loss of vision deserve careful visual field studies and the patient should be warned that the cataract operation will restore only central vision.

R. Grunfeld.

Gjessing, Harald. **A slight improvement of the common capsule forceps.** Acta ophth. **26**:445, 1948.

By filing away the next to the last tooth of the capsule forceps used for removing the anterior capsule in cases of extracapsular extraction a small hole is formed through which the lens capsule herniates as it is being grasped by the teeth. Gjessing removes a sufficiently large portion of the anterior capsule with this forceps to prevent the subsequent development of secondary cataract.

Ray K. Daily.

Huerkamp, B. **Basically different types of lens opacities within the group called "cataracta syndermatotica".** Klin. Monatsbl. f. Augenh., **113**:318-328, 1948.

The cataract of neurodermatitis starts behind the anterior lens capsule, involves the anterior cortex and very slowly and progressively the posterior cortex. Cataract in sclerodermy starts in the posterior cortex and the anterior parts of the lens remain clear. Lens opacities in poikilodermy at the same time in the anterior and posterior cortex as very fine grayish points. Both eyes are involved and maturity of the cataract is reached after days or months whereas the other forms of cataract may take years to reach maturity. The article contains a chart tabulating the most important differential points. (4 figures.) Max Hirschfelder.

Jaeger, A. **Surgery of luxated lenses.** Klin. Monatsbl. f. Augenh. **113**:312-314, 1948.

The author reports the result of lens extraction in 10 eyes with subluxated and totally luxated lenses. Immanent glaucoma was the indication in some. The use of a capsule forceps is recommended whenever possible. Loop extraction was necessary in nine. Two of the three eyes with subluxation had a postoperative vision near 20/100; one had an intraocular hemorrhage. Those with total luxation were much less satisfactory and must be considered poor risks.

Max Hirschfelder.

Mejer, F. **Infection of the lens after perforating injuries.** Klin. Monatsbl. f. Augenh. **113**:366-369, 1948.

Thirteen cases of extraction of infected lenses after perforating injuries are described. Conservative treatment is rarely successful in spite of the newer drugs and fever shock treatment. Whenever the infection is limited to the lens and the anterior part of the globe, a satisfactory result is possible. Four of the author's cases recovered sight after extraction of the infected lens. Max Hirschfelder.

Schott, J., and Dann, S. **Werner's syndrome: a report of two cases.** New England J. Med. **240**:641-644, April 21, 1949.

Two cases of Werner's syndrome are reported. It is an abiotrophic process that first becomes manifest by slow growth during adolescence and graying of the hair at the age of twenty years. Its most striking manifestation is a change in the appearance of the skin which suggests scleroderma but is the result of atrophy of the subcutaneous fat and the underlying muscle. Star-like opacities develop chiefly at the posterior pole and the periphery of the lens. F. H. Haessler.

Thomas, C. I. **Cataract extraction by the suction method.** Arch. Ophth. **39**:805-815, June, 1948.

The author describes a suction apparatus modified after the instrument of Dimitry. The suction method of cataract extraction is used to its greatest advantage when 1. the lens is hypermature, 2. the capsule is tense, 3. the capsule has exfoliated, 4. the lens must be dislocated

before it is tumbled or slid out of the fossa, and 5. the capsule is friable and will not stand any tension. Pitfalls of the operation are discussed and contraindications to its use are pointed out. An analysis of 618 cases of intracapsular extraction is presented to show the comparative frequencies of rupture of the capsule.

John C. Long.

Velhagen, K., Jr. **Surgery of congenital and juvenile cataract.** Klin. Monatsbl. f. Augenh. **113**:305-312, 1948.

Failure in the operative treatment of juvenile cataract is usually due to poor surgery. The author discusses the various complications. He emphasizes the importance of corneal incisions without bleeding. Loss of vitreous is more dangerous in children than in adults. Traction on the zonula is often followed by phthisis. The author recommends simple discission for lamellar cataract in very young children and emphasizes the necessity of doing as small an operation as sule, shrunken cataracts and tough secpossible. Cataracts with thickened capondary membranes should never be pulled out. The procedure results in phthisis bulbi. A so-called double discission is recommended, introducing two small knives from each side through small limbal incisions at the same time and cutting from the pupillary center towards the pupillary periphery. A cut with scissors may sometimes be necessary, but is more dangerous. The posterior lens capsule should not be opened as long as cortical remnants remain in front of the secondary cataract. Linear extraction in one single operation is indicated in older children and young adults only. The author prefers to operate on children with dense lens opacities at the age of 12 months. He discusses the question of early surgery in such cases. (References.)

Max Hirschfelder.

11

RETINA AND VITREOUS

Bedell, A. J. **Diagnosis of retinoblastoma.** Arch. Ophth. **40**:311-316, Sept., 1948.

To illustrate the difficulty of diagnosis, six case reports are given, two of retinoblastoma and one each of angiomatosis retinae, leukosarcoma of the choroid, Coats's disease, and retrolental fibroplasia. Bedell advises against future pregnancy in a family in which more than one child has the disease. One case is not sufficient to condemn the family to a childless existence. Ralph W. Danielson.

Bottasso, G. **A case of retinal embolism of probable venous origin.** Rassegna ital. d'ottal. **17**:384-388, Nov.-Dec., 1948.

A 32-year-old man noted sudden loss of sight in the right eye 11 days after surgery for internal hemorrhoids. There was edema of the macula and in the area between it and the disc, and a sudden marked reduction in caliber at one point in an arteriole in the region. Treatment was ineffectual. It is assumed that a small embolus originated in a hemorrhoidal vein and produced a pulmonary infact from which a minute fragment found its way into one of the arteries to the eye.

Eugene M. Blake.

Carreras Matas, B. **Difficulties in the diagnosis of retinal detachment.** Arch. Soc. oftal. hispano-am. **8**:873-878, Sept., 1948.

A woman, 40 yearrs old, had a central scotoma in the right eye, myopia, negative ophthalmoscopic findings and a recent puerpurium with a dead fetus. The diagnosis of retrobulbar neuritis was made, and treatment prescribed accordingly. Five weeks later the scotoma involved the upper nasal field, and funduscopy revealed a retinal detachment. In

the course of a perforating diathermy-coagulation a small hole was found close to the optic disc. Forty days after the operation the retina was completely reattached, the visual field was normal, and the central scotoma corresponded to the coagulated area. The reasons for missing the diagnosis at the outset are discussed, and it is suggested that an examination of the visual field for blue, and biomicroscopy of the posterior pole of the eye might have helped to avoid the error. In retinal detachment, which disturbs the nutrition of the neuroepithelial layer, the field for blue suffers more than the red-green field. The greater magnification and intensity of illumination in biomicroscopy with a slitlamp might have revealed the retinal detachment. In general, however, this method of examination is more suitable for the study of details in a previously discovered disease than for the search for a lesion. (4 figures.)

Ray K. Daily.

De Berardinis, E., and Bonavolontà, G. **Tissue therapy in ophthalmology.** Boll. d'ocul. **27**:753-772, Dec., 1948.

Among 35 patients with tapetoretinal degeneration and treated with intramuscular cod liver oil injections 26 showed an improvement of variable degree during seven months. The authors feel that further prolonged observations will be needed. K. W. Ascher.

Di Luca, Giuseppe. **Retinal arterial occlusions.** Riv. oto-neuro-oftal. **23**:106-130, March-April, 1948.

Studies of 12 patients from 16 to 76 years of age with unilateral or bilateral occlusion of the central artery of the retina or of its branches by angiospasm or endoarteritis are reported. (References and 10 figures.) Melchiore Lombardo.

Di Luca, G. **Clinical and statistical considerations of periphlebitis retinalis and recurrent vitreous hemorrhages of the juvenile eye and its connections with Buerger's disease.** Boll. d'ocul. **27**:773-788, Dec., 1948.

Di Luca exhaustively studied 11 patients between 18 and 43 years of age with periphlebitis retinae or recurrent vitreous hemorrhages, and all but one showed evidence of tuberculous infection. Twenty patients with Buerger's disease were evaluated as to their ophthalmoscopic findings. Ten of the 20 showed sclerosis of the retinal arteries, 9 had engorged retinal veins, 8 peripapillary edema, 1 an inactive choroiditis, and 4 endarteritis of the retinal arteries. Summarizing his findings and those of other authors he found that among 184 patients with periphlebitis and recurrent vitreous hemorrhages, more than 71 per cent, had tuberculosis, and less than 1 percent showed signs of Buerger's disease. Among 138 patients with Buerger's disease only one had periphlebitis retinae and one recurrent vitreous hemorrhages. The patients with periphlebitis and recurrent hemorrhages belong to a markedly younger age group than those with Buerger's disease. There is no direct relation between th two fundus conditions and Buerger's disease. (References.)

K. W. Ascher.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.

601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month*

MISCELLANEOUS

RESEARCH PROGRAM

Papers presented at the 18th scientific meeting of the Association for Research in Ophthalmology, Inc., Philadelphia, were: "Experimental studies on sympathetic ophthalmia," Dr. Raymond C. Collins, New York; "Correlation of microscopic and slit-lamp examination of developing hereditary cataracts in mice," George K. Smelser, Ph.D., and Dr. Ludwig von Sallmann, New York; "Curare and akinesia for ocular surgery," Dr. John R. Roche, New York.

"Experiments on fatigue of accommodation with asthenopic patients. II," Dr. Conrad Berens, New York, and Saul B. Sells, Ph.D., Randolph Field, Texas; "Some effects of injection of hyaluronidase into the anterior chamber," Dr. Jay G. Linn, Jr., and Lieut. Col. Thomas L. Ozment, Pittsburgh.

"Effects of metabolic poisons and of some other agents on intercellular cohesion in corneal epithelium," Dr. Wilhelm Buschke, New York; "A study of visual mechanism as revealed by the electrical activity of optic-nerve fibers," Dr. H. K. Hartline, Baltimore; "Nutritional supply of corneal regions in experimental animals," Dr. Albert M. Potts and Dr. Lorand V. Johnson, Cleveland.

"Studies on the physiology of the eye using tracer substances: Part III. Further studies on the steady-state ratio of sodium between the plasma and aqueous humor in the guinea pig," Dr. Roy O. Scholz, Baltimore; "Studies of the eye with radioiodine autographs," Dr. Ludwig von Sallmann and Beatrice Dillon, M.A., New York; "The cyanide sensitivity and cytochrome-C content of the crystalline lens," Dr. L. O. Ely and W. A. Robbie, Ph.D., Iowa City, Iowa.

"A study of the possible conversion of dehydroascorbic acid to ascorbic acid in the aqueous humor," V. Everett Kinsey, Ph.D., Boston; "The histochemical localization of cholinesterase," George B. Koelle, Ph.D., and Dr. Jonas S. Friedenwald, Baltimore; "Polysaccharides in ocular tissue," Dr. Robert Day, Baltimore; "Problems in the histochemistry of the eye," Dr. Jonas S. Friedenwald, Baltimore.

"Beta-ray application to the eye with description of an applicator utilizing SR^{90}," Dr. H. L. Friedell, Dr. C. I. Thomas, and J. S. Krohmer, M.S., Cleveland; "Beta irradiation: An evaluation of a radium-D applicator for ophthalmic use," Dr. Fred M. Wilson, Chicago; "The effect of low voltage roentgen rays on the normal and vascularized rabbit's cornea," Dr. Harold G. Scheie, Dr. Richard H. Dennis, Dr. Richard C. Ripple, and Dr. Larry L. Calkins, Philadelphia.

SECTION ON OPHTHALMOLOGY MEETING

At the meeting of the Section of Ophthalmology, American Medical Association, Atlantic City, June 8th to 10th, the following program was given:

Chairman's address by Dr. M. Hayward Post, Jr., Saint Louis, and a symposium on ocular injuries conducted by Col. William Stone, Washington; Dr. W. Morton Grant, Boston; Dr. David G. Cogan, Boston; Dr. Brittain F. Payne, New York; Dr. Don Marshall, Kalamazoo, Michigan; Dr. Alston Callahan, Birmingham; and Dr. Derrick Vail, Chicago.

"A clinical study of 200 cases of retrolental fibroplasia," Dr. Merrill J. King, Boston, with Dr. William C. Owens, Baltimore, opening the discussion; "Retrolental fibroplasia: A study of its pathology," Dr. Parker Heath, Boston, with the discussion opened by Dr. Algernon B. Reese, New York; "Prophylaxis of gonorrheal ophthalmia of the newborn," Dr. James H. Allen and Dr. Luciano Barrere, Iowa City, Iowa, with Dr. Alan C. Woods, Baltimore, opening the discussion.

"The frequency of the occurrence of cataract in atopic dermatitis," Dr. Alfred Cowan and Dr. Joseph V. Klauder, Philadelphia, with Dr. William P. Beetham, Boston, opening the discussion.

"New trends in ciliary-body surgery for the relief of glaucoma," Dr. Gambiattista Bietti, Pavia, Italy, with the discussion opened by Dr. Derrick Vail, Chicago; "Flicker fusion field: III. Findings in early glaucoma," Dr. Paul W. Miles, Saint Louis, with Dr. Lawrence T. Post, Saint Louis, opening the discussion.

"General anesthesia for cataract surgery," Dr. H. Douglas Sanders and Dr. Norman L. Cutler, Wilmington, Delaware, with Dr. John H. Tucci, Boston, opening the discussion; "The age norms of refraction and vision," Dr. Felician J. Slataper, Houston, Texas, with the discussion opened by Dr. Alfred Cowan, Philadelphia.

"The syndrome of aneurysm of the intracranial carotid: Frontal headache with oculomotor nerve paralysis," Dr. Rudolph J. Jaeger, Philadelphia, with Dr. Frank B. Walsh, Baltimore, opening the discussion; "The use of curare in intraocular surgery," Dr. Daniel B. Kirby, New York, with Dr. Frederick C. Cordes, San Francisco, opening the discussion.

* To receive adequate publicity, all notices of meetings should reach the editor three months in advance of the meeting date.

Section exhibits

Among the ophthalmic exhibits shown in the Scientific Exhibition at the A.M.A. convention were: "Hydrodynamics of the aqueous veins," Dr. K. W. Ascher, W. M. Spurgeon, and Jean Schuff, Cincinnati; "The corneal lens: Theory and application," Dr. M. W. Nugent and Dr. Kevin M. Tuohy, Los Angeles; "Pathology of the eye: New technique for preparations," Dr. Brittain F. Payne, Dr. Joseph H. Krug, and Dr. Edgar Burchell, New York; "Pathology of the eye in old age," Dr. A. L. Kornzweig, New York; "Office bacteriology of the eye," Dr. Frederick H. Theodore, New York; "Kodachrome studies of the ocular fundus," Dr. Dan M. Gordon, New York; "Cerebral angiography related to ophthalmology," Dr. Donald J. Lyle and Dr. Frank H. Mayfield, Cincinnati; "Glaucomatous excavations," Dr. Peter C. Kronfeld, Dr. Roy O. Riser, and Dr. John T. Parker, Chicago.

Dr. Georgiana D. Theobald, Oak Park, Illinois, Dr. Edwin B. Dunphy, Boston, and Dr. Phillips Thygeson, San Jose, California, comprise the exhibit committee of the Section on Ophthalmology.

Societies

Reading program

Dr. S. Gordon Castigliano, Philadelphia, spoke on "The oral cavity; the physician and the dentist," and Dr. Benjamin F. Souders, Reading, spoke on "Ocular manifestations of tropical diseases and their treatment," at the 93rd regular meeting of the Reading Eye, Ear, Nose, and Throat Society, May 18th, held jointly with the Reading Dental Society.

Milwaukee society honors Dr. Haessler

The May 24th meeting of the Milwaukee Oto-Ophthalmic Society was a testimonial dinner for Dr. F. Herbert Haessler in honor of his appointment as clinical professor of ophthalmology, Marquette Medical School.

Dr. Haessler, abstract editor of the Journal, gave up his private practice last January in order to devote all of his time to the teaching of ophthalmology.

Among the speakers for this occasion were: Dr. Gustave Guist, professor of ophthalmology, University of Vienna, who was special guest speaker; and Dr. John Hirschboeck, dean, University of Marquette Medical School, who spoke on "Plans and objectives in ophthalmological teaching at the Marquette Medical School."

Preceding the induction of Dr. Haessler to honorary membership in the society, the annual meeting and election of officers took place.

Personals

Gives Montgomery Lecture

Dr. Daniel B. Kirby, New York, gave the Montgomery Lecture at Trinity College, Dublin, on May 12th. The subject of his address was "The preparation of the whole patient for cataract surgery." On May 23rd, Dr. Kirby presented a paper on cataract surgery before the French Ophthalmological Society in Paris.

To lecture in Mexico

On August 15th to 27th, Dr. Brittain Ford Payne, clinical professor of ophthalmology, New York University Medical College, surgeon and director of pathology, New York Eye and Ear Infirmary, New York, will give a series of lectures and demonstrations on the "Histopathology of the eye," in Mexico City.

Among the subjects to be discussed by Dr. Payne are: "Surgical anatomy and histology of the human eyeball," "Microscopic anatomy in detail," "Normal crystalline lens," "Diseases of the cornea, sclera, and anterior segment," "Granulomas of the eye," "Glaucoma and cataract," "Traumatic lesions of the eye," and "Intraocular neoplasms."

Announcement

Home study courses

Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1st and continue for a period of 10 months. Registrations must be completed before August 15th. Detailed information and application forms may be obtained from Dr. William L. Benedict, executive secretary of the Academy, 100 First Avenue Building, Rochester, Minnesota.

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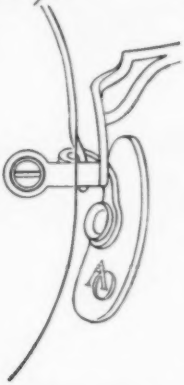

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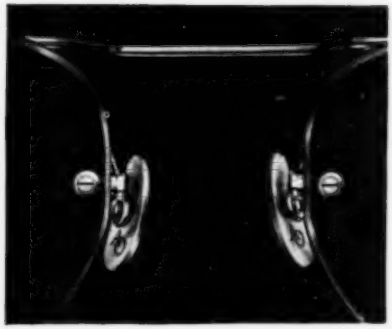

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